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HYPERTROPHIC OSTEOARTHROPATHY: WITH REPORT OF TWO CASES.[1]

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The small number of cases of this condition hitherto published in this country (nine in all), rather than its clinical importance, must be my excuse for reporting my observations on two patients who presented in differing degree the striking picture of the affection. It is important in any unusual or newly differentiated morbid condition that sufficient observations should be recorded to afford a secure basis for generalization and interpretation. In the present instance the name most frequently applied to it is sufficient proof of the foregoing statement. Marie's original publication in 1890 was entitled "De l'osteoarthropathie hypertrophiante pneumique," because, in the cases which he described, the change in the bones had seemed secondary to lesions of the lungs. Subsequent cases have shown conclusively that this connection is by no means constant. As early as 1889, and again in 1891, in an article written before the appearance of Marie's, v. Bamberger discussed eleven cases of a similar nature under the heading "Knochenveränderungen bei chronischen Lungen- und Herzkrankheiten," but the lack of a new definitive appellation left Marie's unwieldy and, in some cases, inapplicable name the field, and the bulk of recorded cases will be found under hypertrophic pulmonary osteoarthropathy. Arnold, shortly after, proposed secundäre hyperplastische ostitis as a substitute; Massolongo, followed by Thayer, secondary hypertrophic osteoarthropathy; and Sternberg, writing in Nothnagel's *Specielle Pathologie und*

[1] Read before the Section on Medicine, New York Academy of Medicine, May 19, 1903.

Therapie, toxigene osteoperiostitis ossificans. To the latter name the objection can be urged that, at least in some cases, the toxigenic origin is not demonstrable. Inasmuch as the bulk of the literature on the subject is now indexed under osteoarthropathy, it seems wise to me to retain this distinctive name, even though Freitag objected that all cases do not show joint lesions, and omit all uncertain qualifying adjectives; though, for the sake of the medical student and of one's own power of articulation, wishing it might be otherwise.

The essential lesions of the condition produce such characteristic changes in the contour of the extremities and are so well defined in radiographs, that I will first record the important features of my own cases and call attention to the accompanying pictures, returning to the general consideration later.

Case I. — J. T., aged twenty-two years. Single. American. Formerly employed in out-door labor. Father died of tuberculosis. Drank and smoked up to one year before. No previous illness. Came to University and Bellevue Hospital Medical College Clinic February 26, 1902.

Present illness began with a cold six years before. For two years this persisted without much annoyance until suddenly one day, without known cause, he coughed up a large amount of purulent matter. There was no pain or dyspnœa accompanying this, no acute illness, accident, inhalation of foreign body, nor disturbance of general health.

From that time on this sudden raising of large quantities of purulent sputum recurred at regular and diminishing intervals; for the first year once a day, of late four or five times a day. Twice in the last two years he had been in bed with fever, etc., called pneumonia. For one year the sputum had been distinctly fetid and at times streaked with blood. While being examined he coughed up about eight ounces of greenish pus with an exceedingly fetid odor, which streamed out of mouth and nose. For two years sweats at times. For the last two years he had noticed progressive enlargement of his hands and feet.

Physical Examination. Height about five feet eleven inches; somewhat emaciated; color sallow. Weight one hundred and fifty-two pounds. One was struck with the tremendous size of the hands and feet; the finger ends clubbed, with characteristic curving of nails; color somewhat cyanotic. Face showed none of the changes of acromegaly, and the hands were of a different type. The lower third of leg and arm were much enlarged, so that both had a nearly straight outline from knee and elbow to ankle and wrist. This was evidently due in part to increase in the soft tissues and in part to marked thickening of the bones. There was no pitting. Pulse 90. Temperature normal.

The chest showed marked dulness over lower part of right lung, which in fourth space anteriorly changed to a cracked-pot note after raising the abundant secretion mentioned. Over this there were cavernous respiratory murmur and voice. Elsewhere over the dull area feeble respiratory murmur and voice, and abundant coarse, liquid râles. At the left base there were scattered coarse râles. The heart was normal, the liver and spleen not enlarged. Fluoroscopic examination showed a heavy shadow over right lower chest. Urine negative.

Sputum fetid, purulent, contained diplococci and bacilli in large number; no tubercle bacilli on two examinations; a few Charcot-Leyden crystals.

The condition was evidently one of bronchiectasis with fetid bronchitis and secondary hypertrophic osteoarthropathy. Radiographs made at this time show very clearly the irregular deposition of periosteal new bone on the shafts of tibia, fibula, radius, and ulna, with much thickening of their lower third. The bones of the hand and foot show little change.

An operation for drainage of the bronchiectatic cavities was consented to. On March 26th Prof. B. F. Curtis opened the thorax over the area of cracked-pot resonance. The pleura being found free, it was decided to set up adhesions and open the lung at a second operation. Recovery from chloroform was good, but unfortunately the patient could never be brought to consent to the subsequent step and left the hospital.

On May 23d the accompanying photographs were taken. At that time the enlargement of the distal extremities of arm and hand were more pronounced in contrast to the general emaciation. A detail of the measurements taken at this time would be of no value, but the following series emphasizes the picture:[1]

Forearms.		*Right.*	*Left.*
Length.	Int. condyle to styloid	26 cm.	26 cm.
Circum.	10 cm. below condyle	23 "	22 "
"	15 cm. " "	20.5 "	19.5 "
"	20 cm.	21 "	19.5 "
"	1 cm. above styloid	21 "	19.8 "
"	of hand around knuckles . . .	21.5 "	21 "

The measurements show very sharply the loss of the normal taper toward the wrist in the contour of the forearm. At this time the patient developed considerable enlargement of the knee-joints, without pain. This was in part due to an actual effusion into the joint. At no time was spontaneous pain in the bones marked.

During the fall of 1902 his condition was unchanged, but on January 4th he became very ill, with fever, dyspnœa, increase in amount and fetor of the sputum, and died January 22d. Unfortunately he lived on Long Island, and his death was not reported to me until after the funeral; so no autopsy was obtained.

Case II.—S. B., aged twenty-eight years. Married. Polish. Tailor. Family history negative. Temperate habits. Came to University and Bellevue Hospital Medical College Clinic May 21, 1902.

Present illness began eight years before with pain in right side and hard, continuous cough. From the start he says that he raised much mucopurulent sputum with a very fetid odor. He was in bed at home seven months and became much emaciated, but in the summer he improved and went back to work. The next winter he was in the Eastview Hospital, Westchester Co., for eight months, working again in the summer. Since that time there has been the same recurring cycle, sick

[1] In Thayer's Case IV. the corresponding measurements were: circumference, 1 cm. above styloid, 17 cm. on each side, the length of forearm being the same, 26 cm.; Marie's original Case B showed a circumference at the level of styloid of 23 cm.; normal wrist measure would be about 16 cm.

FIG. 1.

CASE I.—Radiograph of forearm and hand.

FIG. 2.

CASE II.—Radiograph of forearm and hand.

most of the winter and comparatively well in summer. His weight in summer reaches one hundred and seventy-five pounds. For five years he has had bloody expectoration for three or four days, at intervals of about four months. For six years patient had noticed increasing size of his hands and feet and at times spontaneous pain in legs. No history of inhalation of a foreign body could be elicited. His complaints were of pain in the right chest, restriction of respiratory movements with some dyspnœa on exertion, and cough with expectoration on rising in the morning.

FIG. 3.

CASE I.—Arm and hand.

FIG. 4.

CASE II.—Arm and hand.

Physical Examination. Height five feet eight and three-quarter inches. Weight one hundred and eighty pounds. Healthy color. Hands markedly enlarged in all measurements; finger ends clubbed. Nails very large, curved, color slightly cyanotic. Feet show some changes in a less degree.

The ankles and wrists seem definitely, though slightly enlarged, without œdema or increase in soft parts.

Measurements of forearm and hand.	*Right.*	*Left.*
Length	27–1.2 cm.	27 cm.
Circumference 7 cm. above wrist	19 "	19 "
" at wrist	18.5 "	18.5 "
" of hand about knuckles	22 "	22 "

The chest showed a circumference of 18½ inches right, 18 inches left. Heart normal, not displaced. Lungs: right, marked dulness below fourth rib anteriorly, with slight dulness a space higher. Posteriorly, marked dulness in right interscapular region, with some dulness in suprascapular and infrascapular regions. Over the interscapular region were cavernous respiratory murmur and voice sounds, with coarse, consonating râles. Below, the respiratory murmur was bronchovesicular and the râles smaller. Above and anteriorly were coarse mucous râles, without marked change in respiratory murmur or voice. The left side was normal.

Fluoroscopic examination showed a deep shadow in the right interscapular region, with a lighter shadow below. Two examinations of sputum failed to show tubercle bacilli. The diagnosis seemed probably fibroid induration of the right lower lobe, with bronchiectatic cavities, perhaps secondary to the condition of fetid bronchitis and bronchiectasis, though a tuberculous origin could not be excluded; secondary hypertrophic osteoarthropathy.

The latter condition, as shown by the photographs and measurements, was not nearly so pronounced as in Case I. The radiographs show some irregular osteophytic outgrowths on the shafts of radius and ulna, less on tibia and fibula. The general contour of the bones is preserved.

This patient when last seen, a few months ago, was in the same condition. There have been no joint swellings at any time, though a little pain in the joints.

These cases illustrate sufficiently well the symptomatology of Marie's and v. Bamberger's types of the affection. Both have the great enlargement of hands and feet, with the clubbed fingers and curved nails. The latter shows, in addition, a moderate thickening of the distal ends of forearm and leg bones. In the former this is much more pronounced, involves probably the whole length of the shaft in some degree, and affects also the other long bones, and there is a conspicuous swelling of the joints, best appreciated in the knees. My two cases differ from the classical descriptions only in their comparative freedom from pain in the bones involved.

Pathological Anatomy. Two distinct sets of changes are found at autopsy in this condition, one involving the bones, the other the soft parts. The bone lesion is practically described by the name osteoperiostitis ossificans. There is a chronic inflammatory process which leads to rarefaction of the central cancellous bone and to the production of layers of osteophytic growth at the periphery (Lefebvre, v. Bamberger). In the ordinary cases this has involved especially the lower third of radius and ulna, tibia and fibula, to a less extent the carpal, metacarpal, and proximal phalangeal bones, and the lower end

of humerus and femur. The terminal phalanges show very little thickening. In the extreme cases, such as the skeleton pictured by Sternberg, there is marked irregular thickening of all bones, and the spinal column shows kyphosis and scoliosis. Histologically, round-cell infiltrations and thickening of the vessel walls have been found (Sternberg). Chemical analysis shows too high a proportion of magnesium salts and of fat (Lefebvre).

In the joints Thorburn found some excess of fluid and erosions of

FIG. 5.

CASE I.

the cartilages. The soft parts show a marked increase, especially in the terminal phalanges, and to this the Hippocratic fingers are due. Buzzard found the increase of subcutaneous fat most noticeable; Hirschfeld considers the hypertrophy as affecting chiefly the skin. Freytag saw great distention of the capillaries in the bed of the nail.

The two questions which concern us at present in the discussion of this obscure process are:

1. What shall we include under the term hypertrophic osteoarthropathy?

2. What is the real cause of the pathological changes in the bones and soft parts?

1. *What shall we include under the term hypertrophic osteoarthropathy?* Of cases clinically resembling the original descriptions of v. Bamberger and Marie, there can be no question. A glance at the literature, however, shows two classes of a different kind reported under this name. These are the cases with similar changes in the bones and soft tissues of the extremities, but in whom no primary dis-

FIG. 6.

CASE II.

ease existed previous to the onset of the deformities (see analysis of cases), and, on the other hand, the ordinary clubbed fingers met with in chronic heart and lung affections. The first class, I think, from the identity of the lesions, must be admitted as hypertrophic osteoarthropathy, even though the term secondary does not seem to apply. As to the clubbed fingers, however, there is much discussion. Freytag excluded them from consideration. Lefebvre, however, considered them phenomena of the same order and was misquoted by Sternberg, who also classes them as the mildest type of the condition, thinking it best

to group all the material from a clinical standpoint until our knowledge of the actual etiology is greater.

Before many autopsies had been recorded, and while radioscopy was unknown, it was supposed that the clubbed fingers in osteoarthropathy depended upon thickening of the phalangeal bone. This, however, has been proven false, almost the whole size being due to hypertrophy of the soft parts. The recent observations of Schittenhelm are important in this connection. In an advanced case of osteoarthropathy of Marie's type he saw, between May and July, 1901, moderate diminution in the size of the hands. Careful measurements of radiographs during this period demonstrated that the retrograde change involved the soft tissues almost exclusively. A fourteen-day metabolism experiment during the same period, with known P: N ratio in the food, showed normal phosphorus excretion in the urine.[1]

Other instances of improvement in the condition of the extremities with cure of the primary disease have been reported by Godlee and Mettenheimer (empyema), and Schmidt (syphilis); and in simple clubbed fingers secondary to empyema Gillet and Moizard saw their complete disappearance and Moussons their marked diminution.

In the light of the very evident clinical relationship, it therefore seems wise to me to consider the conditions as different stages of the same process until a case is found with the bone lesions and no clubbed fingers, or until a certain and different etiology is proven for the two manifestations. It is probable that careful radiographic observation of the distal ends of radius and ulna might show slight thickenings in many cases of simple Hippocratic fingers. Von Bamberger's cases 8, 9, and 11 were clinically of this type, the slight osteophytic growth being found first at autopsy.

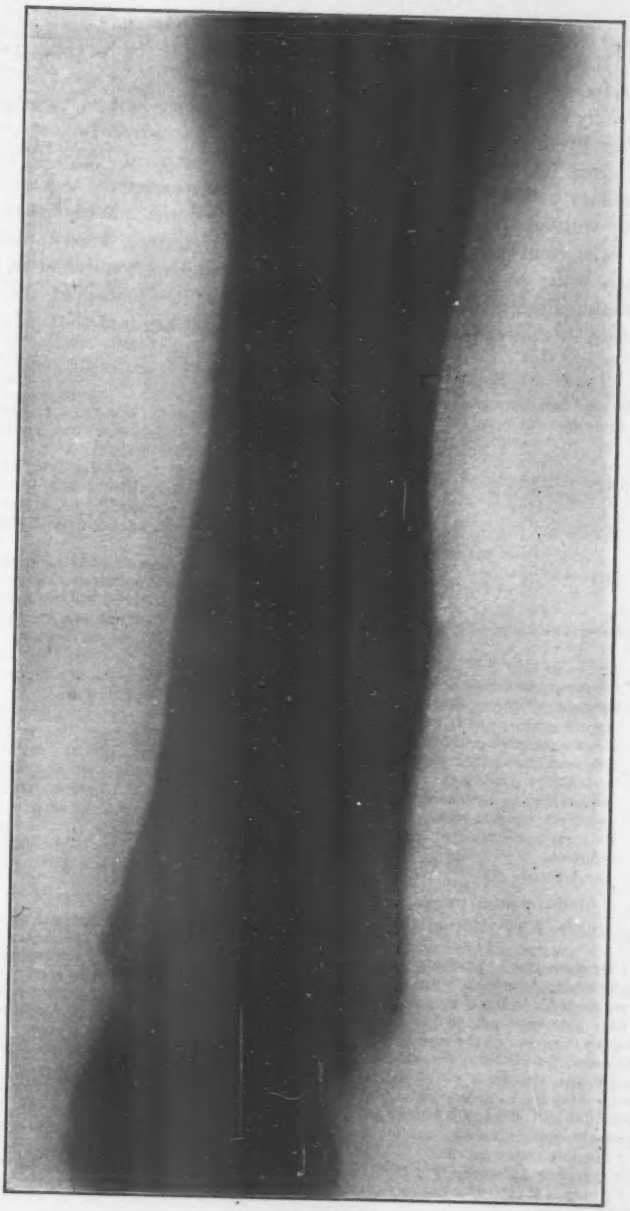
2. *What is the real cause of the pathological changes in the bones and soft parts?* No certain answer can be given to this question to-day, and we can only consider the theories advanced in the light of our present knowledge. As these are largely based upon the association of the symptom-complex with certain primary lesions of other organs, a grouping of the recorded cases under these primary diseases must be made. Teleky, in 1897, made the following classification of causative conditions:

1. Diseases in which suppurative or gangrenous processes occur: Tuberculosis of the lungs (with cavity formation), bronchiectasis, empyema, pyelonephrosis, dysentery. The most common causes.

2. Infectious diseases and chronic intoxications: Pneumonia, pleurisy, influenza, syphilis, chronic jaundice, alcoholism.

[1] This is the only trustworthy metabolism experiment in osteoarthropathy, Guérin and Étienne, who claimed to have found increased Ca and diminished P in the urine, having failed to estimate the amount in food or feces.

FIG. 7.



CASE I.—Radiograph of leg.

3. Valvular heart lesions, especially congenital.

4. Malignant tumors: Sarcoma and carcinoma of the lungs; parotid sarcoma.

Teleky added a fifth group, diseases of the nervous system, especially syringomyelia, but the bone changes here are of a different nature.

Thayer, in 1898, gave a classification of 55 cases, in which 43 were secondary to disease of the lungs and 12 to other causes.

As neither of these authors has so arranged the material that we can refer from their classification to the original sources, it has appeared worth while to me to group all the cases which I could find in the available literature to January 1, 1903, giving the names of the reporters, so that any future reclassification might begin at that point.[1]

TYPES OF MARIE AND V. BAMBERGER, NINETY-THREE CASES.

The condition followed:	*Cases.*	
Bronchiectasis	20	v. Bamberger, 7; Doebbelin, 2; Freytag, Godlee, Massolongo, Sitta, Thayer, 4; Walters, Janeway, 2.
Pulmonary tuberculosis with cavities	14	v. Bamberger, 2; Fraentzel, Hirschfeld, Lockwood, Mettenheimer, O'Carroll, Packard, v. Recklinghausen, Teleky, Therese cit. by Lefebvre, Thorburn, 2; Whitman.
Empyema	11	Davis, Demons et Binaud, Godlee, 3; Lefebvre, Prokop a Stretti, Rauzier, Sollier,[2] Springthorpe, Villard.
Chronic bronchitis	6	Edgar, Field, Gerhardt, Gillet, Stembo, Sternberg [also pleural effusion].
Abscess of lung	1	Kerr.
Pulmonary tuberculosis, no cavities	1	Mestre.
Pulmonary tuberculosis and spinal caries	1	Thorburn and Westmacott.
Chronic pleurisy and spinal caries	1	Orillard.
Chronic pneumonia	1	Schittenhelm.
Retracted chest after empyema (?)	1	Rendu et Bulloche.
Hydatid cyst (?) of lung	1	Thoinot et Delambre.
Caseous mass in right lower lobe; arteriosclerosis; cysts in brain	1	Waldo.
Malignant growths of the pleura:		
Sarcoma	4	Elliot,[3] Hasbrouck,[3] Saundby, Virchow.
Carcinoma	2	Ewald,[3] Norrie.[4]
Total associated with chronic disease of lung or pleura	65	

[1] My references undoubtedly cover the bulk, though not quite all, of the recorded cases. To avoid any discussion I have arranged separately all cases in which only the clubbed fingers were present. In the celebrated cases of the brothers Hagner (Fredreich, Erb, Marie, Arnold) I have not considered the diagnosis secure, and have not included them. I also agree with Walters in excluding the cases of Murray and Verstraeten and that of Legrain quoted by him. My reason for including the other cases which were not proven secondary I have given elsewhere. An abstract of all cases reported to 1895 may be found in Walter's paper on "Osteoarthropathy and its Relationships," which comparison with the originals has proved accurate. A clinical and radiographic study of six cases by Reynaud and Audibert I have been unable to obtain, so their cases are not included.

[2] Possibly syringomyelia.

[3] Also pleural effusion.

[4] Unreported case with autopsy at City Hospital, New York, by kind permission of Dr. Van Horne Norrie.

	Cases.	
Chronic jaundice in hypertrophic cirrhosis.	8	Gilbert et Fournier, 2; Obermayer, 5; Parmentier, Castaigne.
Syphilis	3	Chrétien, Schmidt, Smirnoff.
Valvular heart lesions (uncomplicated)	2	v. Bamberger, Cases 10 and 11.
Ulcerated carcinoma of œsophagus	1	Hirschfeld.
Sarcoma of parotids	1	Clément et Posmantir, cit. by Kalindero.
Chronic dysentery	1	Teleky.
Chronic alcoholism; pneumonia	1	Joffroy.
Acute infectious disease: pneumonia	1	Marie.
influenza	1	Redmond.
Total associated with other diseases	19	
No preceding cause:		
Patient otherwise healthy	5	Decloux et Lippmann [Lemercier same], Gessler, Salles et Halipré, Stevens,[1] Van der Weidje and Buringh, Boekhoudt.
With subsequent disease:		
Slight bronchitis	1	Gouraud, Marie.
Spinal caries	1	Guerin.[1]
Syphilis, inactive	1	Newton and Mercelis.
Pulmonary tuberculosis	1	Spillman and Haushalter.
Total not proved secondary	9	

CASES WITHOUT ENLARGEMENT OF THE LONG BONES.

The condition followed:	Cases.	
Empyema	6	Bailly, Lefebvre, Chauffard, Moizard, 2; Moussons, Ruehle.
Empyema and pulmonary tuberculosis	1	Gillet.
Empyema and bronchiectasis	1	Marfan.
Bronchiectasis	4	Jovane, 2; Marie cit. by Lefebvre, Möbius.
Pulmonary tuberculosis with cavities	3	Combemale et Chatelin, Hirschfeld, Leon y Avalés.
Chronic bronchitis	3	Leon y Avalés.
Congenital heart lesions	2	Jovane, Variot et Chicottot, cit. by Lemercier.
Immobilization for fracture of wrists, later pulmonary tuberculosis	1	Combemale et Sonneville.
Chronic jaundice	1	Gilbert et Fournier.
Pyelonephrosis and cystitis	1	Marfan.
Ulcerated carcinoma of œsophagus	1	Hirschfeld.
Arteriosclerosis; angina pectoris	1	Vedel.
Leprosy	1	London, cit. by Mettenheimer.
Rachitis	1	Fischer.
Malarial cachexia; cirrhosis	1	Abadie.
Total	28	

These figures are given only because the cases are found in the literature of hypertrophic osteoarthropathy. They are valueless as representing the comparative frequency of clubbed fingers in various conditions, because the common cases in lung and heart lesions are seldom reported. For a careful study of this in a series of cases the reader should consult Freytag's thesis. That clubbed fingers may also occur without known cause is proved by a patient of whom I have personal notes.

A glance at the foregoing figures is sufficient to convince one that the incidence of osteoarthropathy and pulmonary lesions is not to be made light of. The same thing was brought out by Thayer, and certainly negatives the contention of Massolongo that the relation to rheumatic conditions is closer than to lung disease.

[1] Possibly acromegaly.

Another very evident association is with suppurative, gangrenous, and putrefactive processes, Teleky's first class. In this we find 48 of the 93 cases, a considerably smaller number than the 65 in pulmonary conditions.

If we turn to the other end of the list, we see that in 9 cases, 10 per cent. of all recorded, the osteoarthropathy was not clearly secondary to any known disease. With these I think we may place the single cases said to have followed acute pneumonia and influenza, for, were there any real connection with these very common infections, we should see osteoarthropathy with much greater frequency.

The syphilitic cases might also be explained by the hypothesis of coincidence, were it not for Schmidt's patient, who improved markedly under antisyphilitic treatment. The 8 cases secondary to chronic jaundice seem to establish a real connection. The cardiac cases of v. Bamberger, when coupled with the frequent clubbed fingers found in similar conditions, which might also show slight periostitis if studied with the same care, must be included in any attempt at interpretation.

Of the various theories adduced to explain the phenomena, Massolongo's already mentioned, Thorburn's of an attenuated tuberculosis, and the theories of trophic nervous origin have no substantiation. Von Bamberger's original hypothesis, also urged by Marie, that the process is due to the selective action of toxic substances absorbed from the suppurating or putrefying foci, has, with modifications, been the best supported. Sternberg adopts it, considering the periostitis toxigenic. Almost all the pulmonary cases, those following jaundice, dysentery, malignant growths, and syphilis, may be made to fit this explanation, if one is not too specific about the toxic agent and supposes that different poisons may have the same action. Von Bamberger uses the analogy of the effects of small doses of phosphorus on hens, a periostitis ossificans having been set up in this way by Wegner. Thayer, in the same way, compares the condition with amyloid disease, which follows chronic suppurations of bone, more rarely syphilis, chronic malarial cachexia, and malignant disease. This striking association of the one with chronic bone suppuration, of the other with similar processes in the lungs, leads me to question whether the chemical nature of the toxic substance produced in each case may not in some way depend upon a difference in the chemistry of the exudate in the two tissues. That we have to deal with a poison whose constitution is due to metabolic peculiarities of specific organisms associated with the primary lesion seems most unlikely. The absence of accurate knowledge of the pathological chemistry of exudative and necrotic processes makes any theories purely speculative.

Of experimental evidence in favor of the toxic theory we have none. Von Bamberger attempted, by rectal injections of the sputum from cases

of bronchiectasis, to reproduce the lesions in dogs; but, after a six weeks' trial without result, he desisted. So far as I know, no other endeavor has been made along this line.

Besides the toxic factor, however, I think more stress must be laid upon local circulatory conditions than has usually been done. That prolonged venous congestion is the cause of the ordinary clubbed fingers is usually conceded. The most striking instance of its effect is the case reported by Béclère in 1901. The patient developed typical Hippocratic fingers on the right hand, with aneurism of the third portion of the subclavian artery on that side. The pressure on veins was not sufficient to cause œdema or change of color when quiet, but, on use of the arm, the hand became cyanotic. The left hand was perfectly normal.

That stasis also leads to augmented growth of bone is a fact utilized at times in surgery (Krause, etc., cit. by v. Bamberger) This is probably the main factor in the production of the slight periostitis of v. Bamberger's two cardiac cases, as he himself believed. I also consider that it is an accessory one in the advanced osteoarthropathies of empyema, bronchiectasis, lung cavities, malignant growths of the lung, and deformities of the chest due to spinal caries, etc., for, in all these conditions, peripheral stasis is more or less in evidence. Such stasis would lead to a concentration of the circulating toxic products at the distal portion of the extremities and, by making conditions there most favorable for chronic inflammatory changes and bony growth, help to explain the apparent selective distribution of the lesions. In long-standing icterus with hypertrophic cirrhosis the toxic agent must derive its origin from altered metabolic activities, and here, too, circulatory changes may assist.

When we come to the consideration of the apparently primary cases, in some of which the phenomena have been claimed by the patients to have existed since birth and to have been present in other members of their family,[1] we must either beg the question or postulate some peculiar susceptibility of the osseous system to slight toxic influences. That great individual differences in the reaction of the bones to irritants exist in different persons is evident from the varying time required for the repair of fractures.

Two observations remain to be mentioned as bearing on the etiology. Hirschfeld found a marked interstitial neuritis at autopsy in three cases, with extreme enlargement of the hands and feet, two of them also showing the characteristic osteoperiostitis. The pain in these cases had been severe and they showed considerable variation in the size of

[1] Freytag: Two cases, each of which had healthy father with clubbed fingers. Fraentzel: A healthy daughter had clubbed fingers. Decloux and Lippmann: Sister said to be the same. Newton and Mercelis: Healthy brother said to be the same

the members at different periods. He considered this due to vasomotor influences dependent on the neuritis, and proposed the term "dermato-hypertrophia vasomotoria" to describe it. This seems to me one of those "Bandwürmerworten," inveighed against by Virchow, which may be spared. To this interesting finding must be added that of Möbius, who saw, in a case of well-marked clubbed fingers occurring in a patient with bronchiectasis, an extreme enlargement of the ends of the two ulnar fingers follow an ulnar neuritis.

In closing I would urge the necessity for the careful recording of cases of osteoarthropathy and allied conditions by radiographs, that at least the chronological sequence of the lesions may be firmly established. This much may be done by the clinician. The problem of the true etiology, I believe, awaits a larger knowledge of bone pathology and of pathological metabolism than we now possess.

For the radiographs accompanying this article my thanks are due to Mr. E. W. Caldwell, Director of the E. N. Gibbs X-ray Laboratory of the University and Bellevue Hospital Medical College.

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AN HOUR-GLASS STOMACH OBSERVED IN SITU.

By Thomas Dwight, M.D., LL.D.,
Parkman Professor of Anatomy at the Harvard Medical School.

The examination of a series of frozen sections through the trunk of a male subject, which were made during the past winter, revealed a very remarkable hour-glass stomach. Perhaps the description of the organ may be of clinical value, for, so far as I know, this condition had never been observed *in situ;* that is, in a hardened body, as it must be if true ideas are to be gained of its shape and relations. Moreover, this specimen offers suggestions as to the etiology of the condition which if not new have at least been lost sight of, and calls attention to peculiarities of muscular action which at least are not generally recognized.

The body was that of a white man, aged sixty-six years, fairly well nourished. He died rather suddenly, probably of cerebral hemorrhage, about three months after his admission with a septic foot into a pauper institution. There is no record of any gastric symptoms. It appears from the frozen sections that the heart was dilated, especially on the right, and that the tricuspid valve was insufficient. The great veins near the heart were very much distended, but this may have been exaggerated by the venous injection.

The body was injected with a 10 per cent. solution of formalin and frozen. Sections were kept for a week or so in formalin before being

transferred to alcohol. I would say, in passing, with regard to the technique, that could I command at pleasure a temperature of below zero (Fahrenheit), and were the sections once cleaned and mounted never to be touched again, I would have nothing to do with so disagreeable an agent as formalin; but it takes to a great extent the place of cold and adds much to the toughness of the slices, which is very useful if they are to be handled. Fig. 1 shows the general shape of the stomach, being from a photograph of a cast of the interior seen almost directly from above. Figs. 2 to 5 represent the upper surface of four successive slices. Fig. 6 is a view of the cast from the right and from behind. The cast is mounted as nearly as possible in the position it occupied in the body.

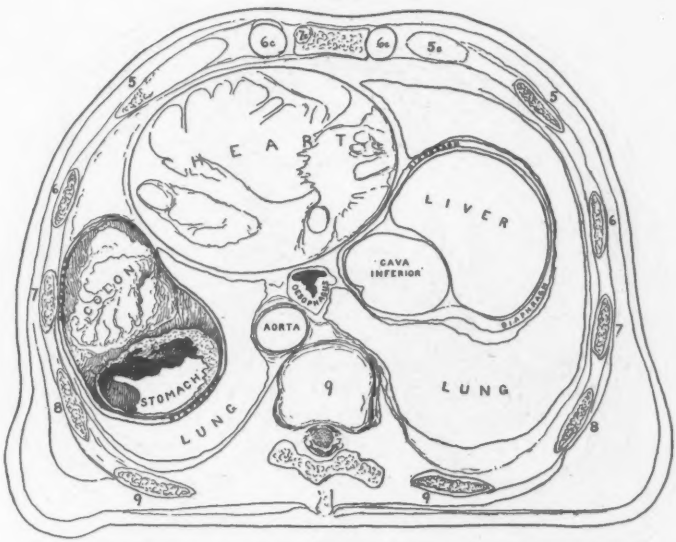
FIG. 1.

Cast of the inside of the stomach, seen almost directly from above. The anterior pouch is at the top; the posterior, with the œsophagus, is below. The black line indicates the median line of the body.

The œsophagus passes the diaphragm opposite the tenth thoracic vertebra in the thickness of the slice, the top of which is shown in Fig. 2, and is seen with the diaphragm still near it at the disk below the tenth vertebra in Fig. 3, running almost horizontally to the left and expanding, trumpet-like, into the stomach (Fig. 6). The mucous membrane does not show clearly the change from the œsophageal to the gastric kind; but this probably occurs nearly opposite the *star* in Fig. 3. The fundus then rises on the left and ends in the thickness of the slice above that shown in Fig. 2, near the top of the ninth thoracic vertebra and a little below the level of the left nipple, which in this body is opposite the fourth intercostal space. This upper division of the stomach (Fig. 2) is nearly oval on section, the anterior and posterior walls being nearer than the borders. Of these walls the former looks decidedly to the left and the latter to the right. The very

summit of this division in the slice above that shown in Fig. 2 was, unfortunately, lost. Having no attachment to the part of the diaphragm just against it, there was nothing to restrain it, and it fell out of place and was overlooked. Happily there were means by which its shape could be approximately determined so as to justify its representation in the cast. The lowest part of the upper division extends downward to opposite the top of the twelfth thoracic vertebra, and then passes into the narrow passage connecting the two divisions. In Fig. 3 we see the upper surface of this narrow passage running forward, expanding and rising, so that in this section a part of each division is shown. The lumen of the constricted portion appears in Fig. 4. Its beginning is best shown in Fig. 3. It is a little behind a transverse

FIG. 2.

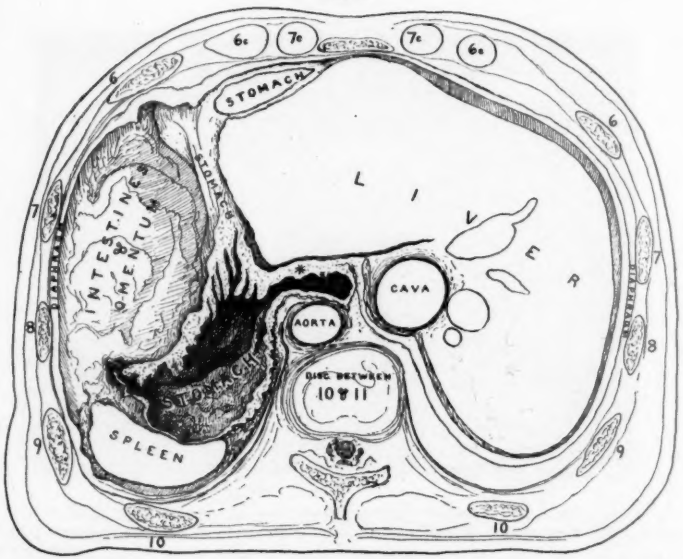


Section at the ninth thoracic vertebra through the upper part of the superior pouch.

vertical plane touching the front of the aorta. The posterior part of the apparent tube shown in Fig. 4 is somewhat behind this and therefore belongs to the first division. The position of the anterior end of the constriction is fairly well shown in Figs. 3 and 4. The measurements of the constricted portion are not very satisfactory, owing to the difficulty of choosing definite limits. The upper border measures about 7 cm. and the lower about 5 cm. The length of the left side may be called 7 cm. and that of the right only 4 cm. Its greatest diameter is from above downward. It is broadest below and would admit a forefinger easily. The very highest part of the inferior division is shown in Fig. 3, opposite the left sixth and seventh costal cartilages near the sternum. This division lies against the abdominal walls in the angle between the eighth costal cartilages, and narrows

gradually to the pylorus without any distinct *antrum pylori*. Fig. 5 shows a part of the pyloric thickening, but gives an erroneous impression of a tubular pyloric portion. The pylorus itself was constricted, but it could easily be dilated to permit the passage of an instrument nearly or quite 1 cm. in diameter. It is placed very far back, within less than one inch of the inferior cava, so that the pyloric portion usurps the place of the first part of the duodenum. The opening is opposite the lower part of the twelfth thoracic vertebra, a little above the plane of Fig. 5, and points directly backward. The general shade and position of the organ are best seen in Figs. 1 and 6. The former gives a good idea of the relative size of the component parts and of the relation of what might be called the long axis of the stomach to

FIG. 3.



Section at disk between the tenth and eleventh thoracic vertebræ opening both pouches and passing above the tubular portion. The star indicates the passage of the œsophagus into the stomach.

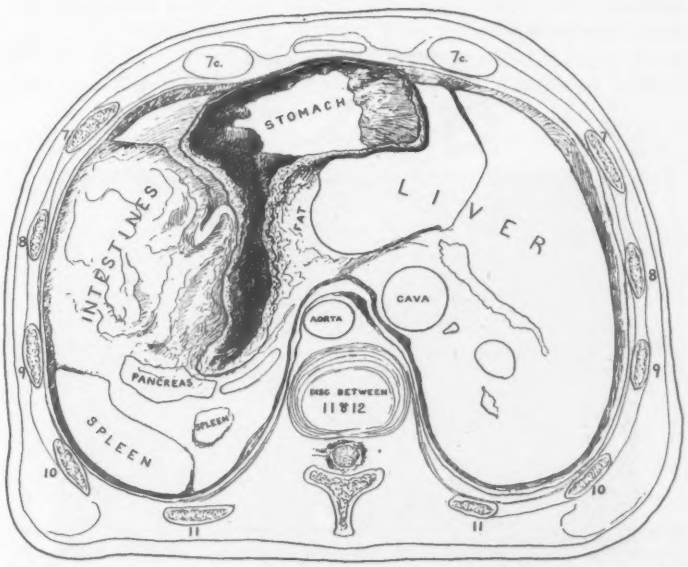
the median plane of the body. The latter shows the general direction of that axis in reference to a horizontal plane.

We may sum up as follows: There are two sacs, of which the inferior is decidedly the larger, connected by a narrow tube. The œsophagus ends nearly horizontally in the superior pouch, which then ascends backward and to the left. The constriction begins at the level of the cardiac opening; it extends forward and somewhat downward, though its under side rises in the middle and empties into the lower or anterior pouch, the top of which is slightly above the level of the lowest part of

the former. The course of the long axis of the stomach may be divided into three parts: The first is downward and somewhat forward, the second almost directly forward, and the third approximately horizontal and transverse. It is worthy of remark that the common axis as a whole might be said to be essentially from behind forward. We shall notice later Birmingham's views on this point.

As to the relations of the stomach, there is not much to say that could not be inferred. The line of the attachment of the great omentum to the stomach is very hard to follow. Apparently it passes behind the fundus in the slice shown in Fig. 2, runs along the left side of the con-

FIG. 4.

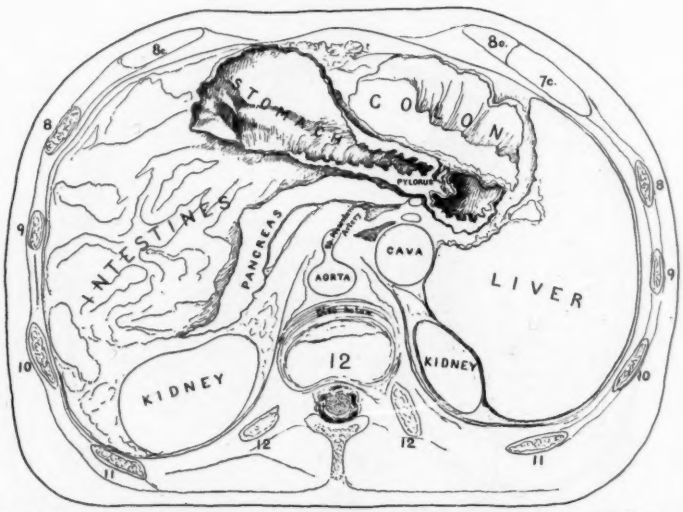


Section at disk between the eleventh and twelfth thoracic vertebræ through the lowest part of the superior pouch, the tubular portion, and the upper part of the inferior pouch.

stricted part, is continued along the lower border of the transverse part shown in Fig. 5, and ends at the right of the pylorus. The course of the lesser omentum is not to be made out. The great omentum, which covers the structures well named by Birmingham the stomach-bed, is not to be recognized. The relations of delicate membranes are not easily followed on frozen sections. The left crus of the diaphragm indents the posterior wall at about the end of the œsophagus. The spleen lies behind and external to the upper pouch. The compensatory relations of the colon with the stomach are well shown. The splenic

flexure rises to the left of the upper pouch. A thick fold of mesentery (probably going to the jejunum) lies against the left of the constricted part, interposing between it and the transverse colon, which reappears beneath the lower pouch. As the latter narrows and passes obliquely backward to the pylorus, the transverse colon lies before it. The bend in the lower wall of the constricted portion is caused by the pancreas, over which it passes. The constricted portion above corresponds very closely to the border of the left lobe of the liver, which is between it and the heart. The second pouch is, of course, covered by the liver, the quadrate lobe covering the pyloric portion. The first portion of

FIG. 5.



Section at the lower part of the twelfth thoracic vertebra, near the bottom of the inferior pouch and just below the pyloric opening.

the duodenum is reduced to next to nothing, the gut descending immediately.

We now come to the most interesting part with regard to the cause of this condition, namely, the walls of the stomach. The thickness of the walls varies very much. They are thinnest at the back of the first pouch, where the wall is probably less than 1 mm. through. The front part of this portion is much thicker than the rest (Fig. 2), and this thickness extends into the constricted portion, being greatest above and on the left. The lower and right sides are less markedly thickened, but they exceed the thickness of the dilated portions. Probably the thickest part of all is where the front of the upper pouch passes into

the top and left of the tubular portion. The beginning of this part is thicker than its end. The thickening of the pyloric part, as shown in Fig. 5, is more apparent than real, owing to the section striking the wall obliquely near its upper part. There is, however, the usual ring at the pylorus.

The mucous membrane is very smooth throughout the lower pouch except for a series of folds in the last inch converging to the pylorus. The upper pouch presents many irregular folds. (It is to be remembered that the very summit, unfortunately, was lost.) The anterior wall of this pouch is covered by a network of folds, to which the cast fails to do justice. These increase in size as they reach the tubular portion, which presents a great abundance of large longitudinal folds, especially in the upper two-thirds of its cavity. They are most developed in the first part of the tube and make a great puckering into the upper pouch (Fig. 3). The highest folds, which are in the constricted portion,

FIG. 6.

Cast of the inside of the stomach, seen obliquely from the right and behind. It shows approximately the inclination of the long axis of the stomach. The star marks the place of the pylorus.

measure 1 cm. or more from base to summit. There are no traces of ulceration or of any disease in any part of the mucous membrane, but the great folds give the impression of the tubular part having been squeezed.

The serous membrane covering the stomach presents in some places a very peculiar appearance. It seems to be filled with minute indentations, which on closer examination often resolve themselves into narrow, longitudinal depressed lines or creases. Sometimes both coexist, and the whole effect is sieve-like. I imagine that tissue like this has been

described as cicatricial, but there is no justification for that term. The peritoneum follows the irregularities of the surface and is quite free from adhesions or other signs of inflammation. This appearance is limited pretty closely to the regions in which the walls are thickened and the folds of mucous membrane exuberant. It descends along the front of the upper pouch onto the top of the first half of the tubular part. It encroaches but very slightly on the second pouch, being found to a small extent on the left of the region shown in Fig. 4, just at its beginning. This portion, however, is insignificant.

Dr. George B. Magrath, of the Harvard Pathological Department, was kind enough to examine microscopically a piece taken from the front wall of the upper pouch a little above the tube, which combined thickness of the wall, redundancy of the folds, and the peculiar appearance of the outer surface. Of course, such a specimen was ill-suited to microscopic examination, as the stomach must have been far from fresh before any hardening agents were used. Dr. Magrath accordingly reported that the mucous membrane showed great post-mortem changes, that glands were to be recognized only here and there, but that the connective tissue between these glands was somewhat hyperplastic, containing more than the ordinary number of cells. The submucosa contained considerable fat tissue, but showed nothing pathological. There was some hypertrophy of the muscular tissue, especially (perhaps solely) of the outer layer, the enlarged aggregations of which gave the surface of the specimen its honeycombed appearance. In a few places there was more connective tissue than is normal between the muscular layers. The serosa showed no signs of thickening in the few places in which it was apparent. I am not aware that a similar microscopic observation has been made on an hour-glass stomach. In spite of its shortcomings, from the nature of the specimen, it is valuable as showing, in addition to an hypertrophy of the muscular tissue, a tendency to hyperplasia by which greater stability is given to the walls.

Among the reasons for which this specimen is valuable may be mentioned the light it throws on the position of the stomach. It is now recognized that the lesser curvature of the stomach is essentially vertical and not transverse; but what is not generally recognized is that the stomach has a very strong forward inclination, and that when empty this may be so great as to make the main axis almost horizontal from behind forward. This is well stated by Professor Birmingham in Cunningham's *Text-book of Anatomy*. He emphasizes also the very important fact that during life hollow organs with muscular walls are not flaccid when empty, but more or less contracted. I have reconstructed another stomach from frozen sections which, without being an hour-glass, was of a shape that would be utterly incomprehensible

to one whose ideas were formed by the older descriptions. It was of small capacity, having been, presumably, nearly empty. It lay nearly horizontally. While the whole organ was in a more or less contracted state, the fundus was puckered up into a separate pouch. Birmingham's dissections of the muscles of the stomach show that there is a whorl over the fundus, which he derives from the innermost layer. My own dissections have inclined me to believe that it is made essentially by the middle or circular layer. As there is more or less intermingling of the layers, it is of little importance which of them predominates; but the contraction of the circular fibres at the periphery of the whorl would account for the puckering of the fundus into a pouch, which is to be observed also in this specimen.

Doubtless there are many causes of hour-glass stomach; in other words, all cases are not of the same nature. We are told that it may be congenital; but, so far as I am aware, there is only one case of a bilocular stomach on record in a fœtus or young child—that of Sandifort[1] in a fœtus. Dr. F. S. Watson,[2] in his excellent paper, classes many as congenital without, as it seems, sufficient grounds. Thus he subdivides all cases into congenital and acquired, the former being marked by the symmetry of the constriction and the absence of pathological processes connected with it. He considers this latter the only positive evidence of its congenital nature. He believes that the acquired cases are generally due to cicatrices from ulcers, or to fresh ulcers, or to adhesions to other organs, etc. He disregards the contractions that occur in the course of digestion. Excellent work has comparatively recently been done on the movements of the gastric walls during digestion by Dr. W. G. Cannon[3] and others, but it is interesting that the occurrence of rings of contraction was known to Sir Everard Home[4] nearly one hundred years ago. He speaks of finding bands in empty stomachs and not always in the same place. Thus they may be at the middle or nearer the pylorus. He insists on the necessity of making the examination soon after death. He made similar observations on animals with simple stomachs. Struthers[5] and others have since alluded to them. Such constrictions can be broken down by more or less pressure of injected air or fluid. Allusion has been made to a constriction becoming fixed or chronic, and this seems to me precisely what has occurred in this instance. The most remarkable feature of this stomach is the length of the narrow part. I am not clear that such another has been described. According to Dr. Watson's classifica-

[1] Obs. Anat. Path., Tome iii. p. 11.
[2] Annals of Surgery, 1900, vol. xxxii.
[3] American Journal of Physiology, 1898, vol. i.
[4] Observations on the Stomachs of Different Animals, etc., Phil. Trans. for the year 1807, vol. xcvii. p. 139.
[5] Monthly Journal of Medical Science, Edinburgh, 1851, vol. i. p. 121.

tion, this must be called congenital; but I do not so consider it. A contraction has somehow become chronic and then consolidated. There is no apparent disease of the mucous membrane, and certainly no inflammation or adhesion of the surface. How this happens I am not prepared to say. No one who has seen the specimen would for a moment admit that any amount of insufflation would overcome the constriction. The fact that it can be overcome in certain cases, but only with considerable difficulty, seems to indicate that there is an intermediate condition between an ordinary transitory contraction and consolidation. There are at least two other places where similar phenomena are observed; one, in fact, is in the same organ. I refer to the occasional appearance of the pyloric portion as a tube with very thick walls. I have observed this also in a series of frozen sections. The appearance of an inch or more of the stomach was that of a piece of small intestine, only with far thicker walls. It was not conceivable that it depended on any transitory contraction of the muscle. It seemed precisely as fixed as in this case. The other region is that of the descending colon, which is sometimes found contracted into a very small tube presenting a series of swellings. This condition may be found associated with irritation of the bowels and also when everything is apparently normal. It is presumably in many cases nothing but the normal contraction of an empty organ with muscular walls; on the other hand, it is sometimes so firm that it is hard to believe it is not permanent. It is further to be said in support of this view that this case was not congenital, but the result of muscular contraction; that in the former hypothesis one would not expect to see the great folds of mucous membrane in the constricted portion, which declare eloquently the effect of constriction.

It has been shown that in digestion waves of constriction pass along the stomach, and are most definite near the pylorus, where, when they become fixed, they no doubt make the single, the double, or even the triple *antrum pylori*, the last of which was mentioned long ago by Retzius.[1] I have seen reason to believe that not rarely there is a slight indication of a narrowing at the middle of the stomach. While it cannot be doubted that hour-glass stomach may be due to ulceration and cicatrices, it is not at all clear how these under ordinary circumstances can cause a circular constriction unless, as is most probable, the irritation produces a contraction of the circular fibres, which become set. Until we see more cases of hour-glass stomach at about the time of birth it is impossible to admit that it is congenital except very rarely. There is no convenient way of accounting for it as a reversion. Indeed, it is much more scientific to ascribe its origin to a

[1] Müller's Archiv, 1857.

phenomenon of contraction natural to a hollow muscular organ in any animal than to have recourse to the doctrine that the exceptional occurrence of some peculiar condition in man is *explained* by its being normal in the black bear or the white whale.[1]

AN OPERATION FOR THE REPAIR OF THE PELVIC FLOOR.

By J. G. Mumford, M.D.,
ASSISTANT VISITING SURGEON TO THE MASSACHUSETTS GENERAL HOSPITAL.

At the present time it seems as though this subject, the cure of ruptured perineum, had been exhausted, because numberless surgeons have devised numberless operations for the repair of the lesion. My object in presenting this communication is to demonstrate an operation simple and effective and applicable to conditions from the most trifling to the most severe.

Long ago Mr. Tait performed an operation by which he succeeded in closing the vaginal outlet without any sacrifice of tissue; but his operation was very limited in scope and was ineffective as a repair of the pelvic floor. Taking Tait's operation as a point of departure I have elaborated a procedure which I have found useful and satisfactory in all routine cases, not only for the repair of the torn perineum, but also for the relief of cystocele and rectocele.

It seems hardly necessary to refer in detail to the anatomy of the region involved, but a few words of explanation are necessary.

The first muscle to be considered is the constrictor vaginæ, which with the sphincter ani goes to make up the rather delicate encircling portion concerned in these lacerations; closely associated with these muscles are the transverse perineal muscles with their attachments to the tuberosities of the ischia. The true pelvic floor, however, is the levator ani, and when this is extensively damaged the most serious displacements follow.

It is obvious that a laceration of these muscular structures not only destroys the inferior supports of the uterus, but of the bladder and rectum as well; especially when the mucous membrane of the vagina is much involved. The prolapse of the bladder and rectum has been

[1] Dr. Bettmann gives a very good bibliography in his prize essay, The Shape and Position of the Stomach, Phil. Monthly Med. Journ., March, 1899. Dr. Watson reproduces in his paper the part relating to hour-glass stomach. I beg leave to point out one inaccuracy and to supply one omission. In the reference to Home's paper the pages are given as 170, 171. The reference on those pages is in an abstract of the papers published in the Transactions. The proper reference is as I have given it. On account of the little-known publication in which it appeared a very excellent paper by Prof. B. C. A. Windle on Sacculation of the Stomach has been overlooked. It is in Proc. Philosoph. Soc. of Birmingham, vol. vi.

happily described as a form of hernia, which, indeed, it is; and various operations have been devised to retain such herniæ and hold these organs in their normal position. The method of all such operations is to restore the injured muscles, and as a preliminary to this the denuding of various parts has been employed. The procedure which I shall describe demonstrates successfully, I believe, that denuding operations are needless. All operations for extensive repairs in the pelvic floor are based upon the need of restoring the continuity of the levator ani muscle. Commonly this muscle is reached and sutured by removing considerable areas of overlying membrane and cicatricial tissue; this involves a tedious dissection in a region which is disinfected only partially and with difficulty, so that the results are never certain and the repair frequently imperfect.

By the method which the following sketches illustrate it will be seen that no tissue is sacrificed, that all the work is subcutaneous, and that the deep parts are quickly and easily reached.

No. 1. shows the first step in this operation, which may be described, for convenience, as a septum-splitting operation. The skin is cut through so as to open a crescentic incision between the vaginal outlet and the anus, and No. 2 shows how the dissection may readily be carried back so as to separate the rectum from the vaginal wall as far up as the posterior cul-de-sac; the finger in the depths of the wound lies against the cervix, no matter what the height of the uterus may be; the outline of the cervix is indicated in the sketch.

This dissection may be done rapidly after some little practice, for after cutting through the superficial cicatrices the high separation may be made with the finger; if care be not taken there is danger of perforating the rectum, but this may be avoided by clinging to the vaginal surface. Even if the rectum be penetrated the resulting wound seldom leads to trouble or interferes with the result. The greatest difficulty, if difficulty there may be said to be, is the sometimes copious bleeding from hemorrhoidal veins. Such bleeding must always be checked, else it leads to hæmatoma and infection; but even in those few of my cases in which the rectum had been perforated no serious trouble has followed. The depths of the wound have, indeed, been infected, some sloughing and abscess formation has occurred, but the integrity of the newly built structure eventually has not been damaged.

No. 3 shows somewhat imperfectly how widely the buried catgut stitches may be carried into the deep parts. The three upper stitches in the sketch are passed through the levator ani fibres and may be sunk freely, and without risk of damage, to any desired depth, the needle being readily carried out to the tuberosities; the two or three lower stitches grasp the severed superficial muscles and the last stitch of all takes up the fibres of the sphincter ani. By this method of stitching

the most extensive lacerations may readily be repaired, as must be obvious from a glance at the illustration.

When these deep stitches are tied the very stout and broad perineal floor is formed, as is shown in No. 4. A second row of buried stitches

is then passed, and finally the skin wound is drawn together, as in No. 5.

It is obvious that the size of the vaginal opening may be made as small as desirable, and in the case of elderly women with extensive herniæ this orifice is made very small indeed.

It would seem that enough has now been done to correct the cystocele and rectocele, and usually this is indeed the case; but if we have to deal with a uterus with elongated cervix and damaged anterior and posterior supports, that cervix may still force its way through the smallest vagina. For such reasons it is sometimes necessary either to amputate the cervix or to fix the fundus of the uterus to the abdominal wall or to perform both operations. In a number of cases in which this complicated operation has been done the functional results have been extremely satisfactory.

In the case of young women in the childbearing period the newly formed perineal body has been found to act in a very normal fashion; it stretches surprisingly before the descent of the fœtal parts, and in the three cases in which I have watched it it has neither proved too resistant nor has it suffered from extensive secondary laceration, as is seen so frequently after the performance of denuding operations followed by labor.

I have a list of forty of these flap-splitting operations, thirty of which were done at the Massachusetts General Hospital and ten in private practice. Of the thirty hospital cases, the immediate results were invariably so excellent that I have been prompted to make this brief report and description of the manœuvre. Eleven of them were in cases of young women and nineteen in women who had passed the menopause. Fourteen have been traced, and the reports from the patients express satisfaction.

Of the ten private cases, three were in young women who have since borne normal children and in a satisfactory manner. The remaining seven cases were those of older persons, who suffered more or less severely from pelvic herniæ. In none of the cases was there done any denuding of the bladder or rectum walls. In four of them the uterus was fixed anteriorly; the anatomical and functional results have been good.

TWO CASES OF LEFT DUODENAL HERNIA, IN ONE OF WHICH THE SAC CONTAINED THE ENTIRE SMALL INTESTINE, THE CÆCUM, AND A PORTION OF THE COLON, WHICH WAS STRANGULATED.[1]

By Leonard Freeman, M.D.,
of Denver, Colorado.

The abdominal viscera and peritoneum are subject to various malformations and malpositions, sometimes acquired and often congenital. They were formerly regarded as mere anatomical or pathological curi-

[1] Read before the American Surgical Association, Washington, May 14, 1903.

osities, but with the growth of abdominal surgery they have acquired a new significance. This is especially true of the peritoneal fossæ and of the irregularities in form and position of the colon, cæcum, and vermiform appendix. Unless the surgeon be familiar with these unusual phenomena his diagnostic ability will be curtailed, and he may at any time be confronted by confusing conditions for which he can find no adequate explanation.

The peritoneal fossæ, for instance, may be responsible for the most remarkable forms of hernia, involving at times the entire bowel, and leading to great perplexity in diagnosis and surgical treatment in case of strangulation. This is particularly true of the duodenal fossæ.

Although the duodenal fossæ were described as early as 1776 by Neubauer, their importance was first pointed out by Treitz in 1857,[1] who described their anatomical peculiarities with accuracy, and emphasized the fact that both small and large herniæ occasionally developed within them.

The duodenal fossæ lie opposite the third lumbar vertebra, where the duodenum emerges from the transverse mesocolon. They are formed by folds of peritoneum attached to the duodenum. If, in the cadaver, the abdomen be widely opened by both longitudinal and transverse incisions, and the colon pulled upward so as to put its mesocolon on the stretch, while the mass of small intestine, with its mesentery, be thrown over to the right, one or two more or less well-defined folds of peritoneum may often be seen fastened to the duodenum near the point where it merges into the jejunum. Rarely are they attached to the jejunum itself. These folds form the borders of corresponding peritoneal fossæ, which were formerly called duodenojejunal fossæ (Treitz); but, seeing that they are more intimately connected with the duodenum than with the jejunum, the term duodenal fossa is preferable.

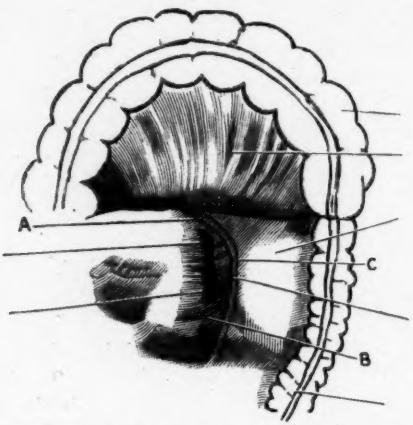
To assist in gaining a clear idea of the situation, one can imagine that the lower portion of the duodenum has been pushed, as it were, through the posterior parietal peritoneum at the base of the transverse mesocolon, dragging with it attachments of the peritoneum or mesocolon, or both, which necessarily form folds. If it is the mesocolon, the fold will have its sickle-shaped concavity toward the pelvis, the resulting fossa extending toward the diaphragm; while, if the posterior parietal peritoneum is concerned, the concave border of the fold will be toward the diaphragm, the fossa bulging toward the pelvis. The former condition gives rise to the superior duodenal fold and fossa (Fig. 1, A), while the latter causes the inferior duodenal fold and fossa, sometimes designated the "fossa of Treitz" (Fig. 1, B).

In addition to these two principal folds, others of minor importance

[1] Hernia Retroperitonealis, Prague, 1857.

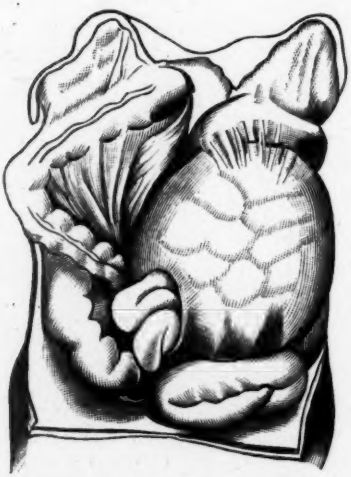
may exist, especially an intermediate one. All of them may be present at once, or any of them may occur singly. They may be so poorly

FIG. 1.



A, superior duodenal fold ; B, inferior duodenal fold ; C, inferior mesenteric vein. (Juvara.)

FIG. 2.



Treitz's case of left duodenal hernia. (Moynihan.)

developed as to escape all but the closest observation, or they may be so prominent as to form deep, well-marked pockets.

The inferior mesenteric vein encircles the left border of the duodenal fossæ, generally passing quite close to it (Fig. 1, C). The left colic artery is usually found more externally. These vessels form the so-called "arch of Treitz." They are a source of danger if an attempt is made to incise the neck of the sac when strangulation is present.

It has always been taught that left duodenal hernia—one in which the sac develops beneath the posterior parietal peritoneum principally to the left of the median line (Fig. 2)—almost invariably occurs in connection with the inferior duodenal fossa; but with this Mr. Moynihan does not agree. He believes that another fossa, which occasionally appears somewhat external to this, is responsible. This he designates

FIG. 3.

Paraduodenal fossa or fossa of Landzert. (Moynihan.)

the "paraduodenal fossa" or "fossa of Landzert." It is formed by a fold of peritoneum (plica venosa), elevated by the inferior mesenteric vessels (Fig. 3). His main argument is based on the fact that the mouth of the hernial sac is always vascular. This argument, however, seems open to question—in the larger herniæ, at least—as it would require but little stretching of the opening to bring the inferior mesenteric vein in such intimate relation with the mouth of the sac that accurate differentiation would be impossible.

Mr. Moynihan, in his excellent monograph,[1] has collected fifty-eight cases of left duodenal hernia, thus showing that, although the affection is sufficiently uncommon, it is not so rare as was heretofore supposed.

[1] Retroperitoneal Hernia, London, 1899.

Strangulation, however, seldom occurs, Jonnesco having found mention of but eight cases in the literature of the subject.

Some time ago it was my fortune to meet with a strangulated left duodenal hernia in a strong, well-developed man, aged forty-seven years, an American by birth, and a miner by occupation. He had always enjoyed good health, except occasional slight indefinite abdominal pains. Following an attack of diarrhœa lasting three weeks he suddenly developed, four days before I saw him, a severe ileus, with the usual symptoms of acute intestinal obstruction. An operation was at once performed. The pulse was then 120 and unsatisfactory in quality, the temperature subnormal, and the capillary circulation poor.

On opening the abdomen we were surprised to find the cavity occupied by an immense tympanitic tumor resembling an ovarian cyst, which could be outlined on each side and below by passing the hand between it and the abdominal walls. The small intestine was nowhere to be seen, but the colon could be felt below and to the right side. After some deliberation we opened the tough, membranous sac, within which was found the entire small intestine, together with the cæcum and some six or eight inches of the adjacent colon. There was also considerable foul and bloody serous fluid, no trace of which existed outside the sac, showing how completely the inner cavity was separated from the outer.

The cæcum, which was distended with fluid feces to the size of an infant's head, and largely gangrenous, lay in the left upper quadrant of the abdomen, just beneath the spleen, in the vicinity of which the swollen appendix was attached by recent inflammatory adhesions. Several inches of the large intestine near the cæcum were also gangrenous. The cæcum and neighboring colon possessed a very long mesentery, which rendered them freely movable.

In tracing the colon from below upward it was seen to emerge from the small pelvis upon the left side, as usual, and then pass directly across the lower portion of the abdomen, coiling itself loosely in the right iliac fossa and right lumbar region, outside the sac containing the small intestine. It then entered this sac through an opening on the right side posteriorly, and, passing beneath a portion of the small bowel, ended in the cæcum in the superior lumbar region on the left side. The colon was strangulated at the point where it entered the sac.

In attempting to relieve the condition it was necessary to resect the gangrenous cæcum, together with some six inches of the large intestine and a considerable portion of the small bowel. In doing this the gut was crushed with an angiotribe, the cut ends invaginated, and the opening closed with an over-and-over suture. A side-to-side anastomosis was then made with a Murphy button between the lower end of the ileum and a loop of colon in the right iliac fossa.

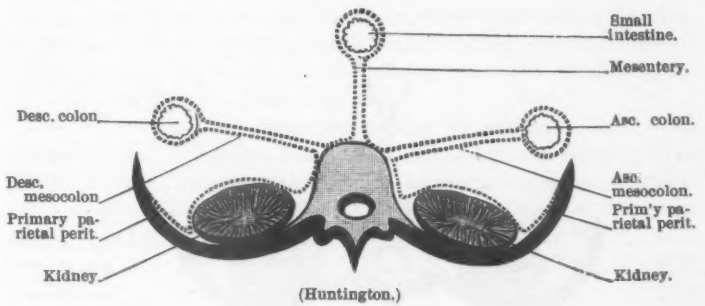
The patient's resisting powers were so poor, and the operation was

so complicated and extended, that death resulted. No autopsy was obtained, but the abdomen had been so freely opened that the condition was evidently as described.

This case seems, in some respects, unique. In no other instance, so far as I am aware, has any portion of the colon been found within the sac of a duodenal hernia; in fact, several writers state specifically that the colon invariably remains outside, as though this were an important characteristic of this form of hernia. This unusual feature is easily accounted for, however, by the mesentery of the cæcum and colon being of abnormal length.

Another anomalous condition is to be noted in the fact that the entire colon lay in folds upon the right side of the abdomen instead of encircling the hernial sac, as was to be expected. Such a malposition is

FIG. 4.



(Huntington.)

usually congenital, as I at first imagined it to be in this instance, and arises from rotation of the umbilical loop during fœtal life in a direction contrary to the normal, thus carrying the whole large bowel over to the right side, beneath the mass of small intestine instead of above it. This, however, is probably not the correct explanation in the present case. It is more likely that the cæcum and colon originally possessed an exceedingly long mesentery (Fig. 4), and that adherence of the descending colon in the left lumbar region failed to take place as it normally should in the course of the development of the individual (Fig. 5). The attachment of such a mesentery would be near the centre line of the body, close to or identical with that of the mesentery of the lesser bowel. Under these circumstances it can be imagined that the duodenal fossæ might be present to even more than the average extent, because of the looseness of the mesocolon, which normally is so firmly adherent to the posterior parietal peritoneum as to practically form part of it.

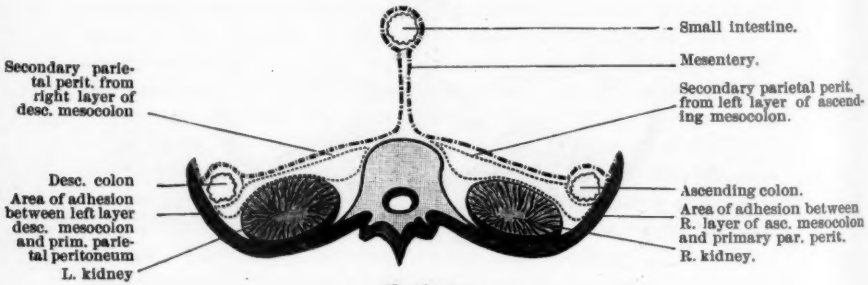
Hence if a duodenal hernia should occur, as it would probably have

an exceptionally good opportunity of doing, the sac would have a tendency to develop to the left of the large bowel instead of to the right of it, thus displacing the descending and transverse colon to the right side of the abdomen, providing the hernia were of large enough size.

In the formation of a postperitoneal hernia such as the one under consideration, the duodenum enters the opening first, dragging after it the jejunum and then the ileum with their mesentery. The cæcum, being attached to the lower end of the ileum, would enter last. As the mouth of the hernia is situated toward the posterior aspect of the sac, the cæcum would be apt to pass beneath the greater portion, at least, of the small bowel, as was the case in the present instance. Strangulation of the colon would be quite likely to occur owing to stretching of the mesenteric vessels and pressure against the sharp edge of the sac.

The fact that the colon alone was found to enter the sac, without any portion of the small intestine being present in the opening, seems

FIG. 5.


Small intestine.
Mesentery.
Secondary parietal perit. from left layer of ascending mesocolon.
Secondary parie-tal perit. from right layer of desc. mesocolon
Desc. colon
Area of adhesion between left layer desc. mesocolon and prim. parietal peritoneum
L. kidney
Ascending colon.
Area of adhesion between R. layer of asc. mesocolon and primary par. perit.
R. kidney.

(Huntington.)

at first a little confusing unless we remember that the duodenum passes beneath the peritoneum to the duodenal fossa, so that when a hernia is formed the remainder of the bowel is dragged through the opening into the space where the duodenum already lies. Hence only that portion of the bowel which has been pulled in, so to speak, can pass through the hernial opening. This condition, which is often found, to say the least, would seem to contradict the opinion of Moynihan that duodenal herniæ almost invariably occur in the paraduodenal fossa beneath the plica venosa, as this would necessitate the entrance of the duodenum into the general peritoneal cavity before it could enter the sac (Fig. 3), which would lead to two sections of bowel being found in the opening instead of one.

Since writing the above I have encountered, during an autopsy, a second case of left duodenal hernia. The patient died from gangrene of the small intestine, accompanied by severe hemorrhage (stomach and

bowels), arising from thrombosis of the mesenteric and portal veins, which in its turn was dependent upon a "hobnail liver." The hernia was apparently in no way concerned with the fatal issue.

The individual was an exceptionally strong and well-developed man, aged forty-seven years, who had always enjoyed good health. He had been subject, however, to indigestion, and occasionally experienced slight pain and uneasiness in the abdomen.

The sac filled the entire left side of the abdominal cavity and pushed well over to the right of the median line. It contained all but six inches of the small intestine, the transverse and descending colon being spread out upon its upper and left outer surface, leaving the ascending colon and cæcum in their normal position. The mouth of the sac was round and smooth and easily admitted three fingers. From it emerged the lower end of the ileum to join the cæcum in the right iliac fossa. The opening was just to the right of the vertebral column, on a level with the crest of the ileum and toward the dorsal and inferior portion of the sac. Its free ventral border was occupied by the inferior mesenteric vein, while the left colic artery ran much further to the left, along the posterior abdominal wall.

The small intestine, which appeared to be unusually short, was arranged in regular parallel folds, like a Mikulicz drain, running from the left below to the right above. The omentum was short and thick and pushed to the right side above the sac. There was no strangulation of bowel or mesentery.

RARE CASE OF APPENDICULAR ABSCESS SITUATED BETWEEN THE LAYERS OF THE MESENTERY OF THE SMALL INTESTINE.

APPARENTLY DUE TO A PERFORATION BETWEEN THE BASE OF THE APPENDIX AND CÆCUM BY A ROUND WORM. FIRST OPERATION, DRAINAGE. SECOND OPERATION FOR OBSTRUCTION IN TWENTY-SEVEN HOURS. ENTEROSTOMY. STARVATION DUE TO HIGH POSITION OF FECAL FISTULA. THIRD OPERATION, SIX WEEKS. INTESTINAL ANASTOMOSIS. RECOVERY.[1]

By Joseph C. Bloodgood, M.D.,
of Baltimore, Md.

I bring this patient before the Society because of a number of rare and interesting features in a very complicated attack of appendicitis. It is the first definite localized abscess which I have

[1] This patient was exhibited before the Medical and Surgical Section of the Medico-Chirurgical Faculty of Maryland, Friday, February 20, 1903.

observed to be situated between the folds of the mesentery of the small intestine, nor previous to this observation have I been able to definitely demonstrate that an appendicular abscess was due to the perforation of a round intestinal worm. In the recent extensive literature on appendicitis I find that these two observations are unique. In the operative procedure (which will be discussed later in detail) necessary to completely drain the abscess and to prevent peritonitis, so much gauze was introduced into the abdominal cavity that one could feel quite certain that obstruction in some part of the lower portion of the ileum would follow. For this reason, twenty-seven hours after the first operation, without waiting for any sign of obstruction except distention, enterostomy was performed through the upper portion of the middle-line wound. At this second operation it was definitely ascertained that obstruction had taken place. Although the enterostomy was performed as low as possible, so much small intestine was excluded that the patient rapidly developed symptoms of starvation, which, in spite of rectal feeding, were extreme by the sixteenth day. The obstructed portion of the ileum did not become patent until all the gauze had been removed from the abscess cavity and until the abscess had practically healed by granulation. This was demonstrated by the introduction of methylene blue about the twenty-eighth day. The patient, however, was then in a condition of such extreme weakness and emaciation that no extensive operation could be attempted to close the fecal fistula. Between the twenty-eighth and forty-second day the fecal fistula was patched up on two occasions. The temporary closure in each instance lasted four to five days. During this time the patient's general condition improved so much that on the forty-second day a more extensive operation with permanent lateral anastomosis was performed. It is now eight weeks since this operation, and you see that the child is in perfect health. Eight weeks ago she weighed twenty pounds; to-day she weighs sixty-five.

The mental condition, due, without doubt, to starvation, was one of great interest. It was fully established by the sixteenth day. For a few days after the enterostomy the child continued to recognize her family and attendant and played with various toys. On about the tenth day she refused to pay any attention to her former almost constant companion, a new doll, and did not recognize anyone. She would not answer questions, and after the sixteenth day hardly ever articulated any sound except a low moan. She picked at the bed-clothes, and if she had not been restrained with a strait-jacket would have removed all dressings. Whenever she was fed or dressed she attempted to bite the nurse or surgeon. The appetite was ravenous. Only on the rarest occasions did she refuse nourishment. The fecal material which was discharged from the upper loop of the enterostomy was usually

well digested. Attempts at feeding through the lower loop failed. Rectal feeding was not well borne. The emaciation was extreme. Twenty pounds on the forty-second day. The child, as a rule, slept fairly well at night; during the day she was restless, with frequent moaning. There was never nausea or vomiting. The examination of the urine was negative. After the sixteenth day, when these symptoms were pretty well established, the patient's general strength remained about the same. To all appearances she had enough intestine to preserve life. This was the most encouraging sign and justified our delay in attempting to re-establish the continuity of the intestine until we were quite certain that the obstruction had been relieved. I was never able to definitely ascertain how much of the small intestine was excluded. I have never previously observed this extreme mental condition due to starvation, and it is interesting to note that this patient received enough nourishment from the upper portion of the small intestine to carry on the function of the lower centres of the brain only. The higher nerve centres were to all appearances inert. The recovery after the anastomosis from both the physical and mental condition of starvation was a very gradual one. Unfortunately, careful notes were not made. According to my recollection, she called me by name for the first time at about two weeks after the anastomosis. It was interesting to note that her memory of previous events should be so good; the probabilities are that she had not seen me a dozen times before she entered the comatose state. To-night you see a perfectly healthy child, without a sign of mental or physical disease and no indication of her illness except the healed abdominal wound. The child weighs, I should say, about ten pounds more than she did previous to her illness. In an older individual we would not expect such a rapid convalescence.

Note, July, 1903. It is now nine months since the operation, and I am informed by Drs. Hildebrand and Yost that the patient is in perfect health, and has exhibited no symptoms of postoperative neurosis or any sign of intestinal obstruction in spite of the extensive postoperative adhesions which must be present.

Clinical History in Detail.

M. K., white, aged eight years. Family and personal history negative. Always a healthy child. I was asked to see the patient in Glen Rock, Pa., by Drs. Hildebrand and Yost. The child had been sick thirty-six hours. There was no history of definite previous attacks, although the child had complained for a number of months, at irregular intervals, of abdominal colic, and had on a few occasions passed a round worm. The present attack was ushered in by severe abdominal pain referred to the umbilical area. This pain had increased somewhat in severity, but had not changed in character or location. Nausea and vomiting were absent; the bowels moved after the use of cathar-

tics. When first seen by her physician last night, twenty-four hours after the beginning of the illness, she was complaining of abdominal pain. On palpating the abdomen they found that all the muscles, chiefly the recti, were held slightly rigid. The child stated that the umbilical area was most tender. The pulse and respirations at that time were but slightly accelerated, and the temperature was 100° F.

I saw the patient on the morning of November 14th, thirty-six hours after the beginning of the illness. The mother told us that the child had passed a very restless night, sleeping but little on account of pain in the abdomen.

Examination. Temperature 101° F., per rectum; pulse 110; respirations 24 to 30, chiefly thoracic. The girl has the appearance of a very healthy child; the muscles are firm; skin of good color; there is very little subcutaneous fat. The face has an anxious expression and the cheeks are slightly flushed, but there is not the drawn or pinched look of a child with peritonitis. The character of the pulse, although accelerated, is good. She complains of constant pain in the middle of the abdomen, and not in the epigastrium or in the right or left side. When the bowels moved during the night there was no pain, and voiding urine gave no discomfort. The abdomen looks but slightly distended; the distention is slightly more marked in the middle zone, beginning a little above the umbilicus and extending to within a few centimetres of the pubes. On palpation both recti muscles are held so rigid that it is impossible to make any depression. This muscle spasm is most marked from midway between the ensiform and umbilicus down almost to the pubes. There is no muscle spasm in either lumbar fossa. Muscle spasm is slight in both iliac fossæ, perhaps slightly more marked in the right. On percussion the liver dulness is not obliterated, nor splenic dulness. The abdominal note is practically normal in the epigastrium and the flanks and left iliac fossa. In the umbilical and upper hypogastric regions extending slightly into the right iliac fossa the percussion note is not flat, but in view of the distention in this area is very much less tympanitic than if the distention was due to bowel. This area of impaired resonance combined with pain and intense muscle spasm associated with fever indicated an acute inflammatory process, probably abscess in the middle zone of the abdomen. There were no symptoms whatever of obstruction. For this reason I was inclined to feel that we should find an abscess, although an unusual position for an appendicular abscess.

Operation. Ether. As soon as the patient was completely narcotized the abdominal muscle spasm disappeared, and one could palpate in the middle zone of the abdomen a mass about the size of the two fists (10 x 10 cm.), slightly movable from right to left, but not from above down. For this reason the abdomen was opened in the middle line from a point a few centimetres above the umbilicus down to the pubes. On opening the peritoneal cavity there was no fluid, fibrin, or adhesions. The small intestines were slightly distended and perhaps the loops over the abscess were a little injected, but there was no peritonitis and no exudate. Beginning at the cæcum and extending at least to the third lumbar vertebra between the folds of the mesentery there was a mass. The exudate at the junction of the appendix and cæcum and ileum was sufficient to obliterate the appendix. Whether the appendix had an unusual position between the folds of the mesentery beneath the ileum

at the junction of the cæcum I could not tell, but at this point there was an exudate of fibrin obliterating anatomical marks. There was no exudate in the mesocolon behind the cæcum. On separating the exudate at the junction of the ileum and cæcum pus was encountered, and a large abscess cavity was found extending toward the vertebral column between the folds of the mesentery.

I considered the possibility of making a lateral incision to the outer side of the cæcum and attempting to drain the abscess from this position, thus avoiding the danger of obstruction, but it could be easily demonstrated that it was impossible. The only way to properly drain this abscess and protect the peritoneal cavity was to use the middle-line incision, pushing the small intestines up and to the left and packing the gauze between the bladder on the lower side and the cæcum on the right. A great deal of packing was necessary to wall off the peritoneal cavity and to prevent leakage of the pus into the pelvic cavity. Although I attempted to use as little packing as possible, I felt quite certain that obstruction would be produced. For this reason I asked Dr. Hume, who assisted me, to remain with the patient and bring her to Baltimore that afternoon or the next morning.

Note on admission to the Union Protestant Hospital, November 15th, 11 P.M., about twenty-four hours after the operation: Temperature 101.6° F., pulse 138, respiration 45. There is a symmetrical distention of the abdomen occupying the umbilical and epigastric area above the gauze drain; the distention is less in the flanks. The liver dulness is slightly obliterated; the child is restless; although the pulse is rapid it is of good character. Since the operation at Glen Rock there has been no nausea or vomiting, and the pulse, which was 178 after the operation, has varied between 130 and 140; the respirations from 24 to 32. The child complained of some abdominal pain, but slept at intervals during the night. From the clinical picture at this time one could not be certain that there was definite obstruction; distention of the degree present is frequently seen after laparotomy. I felt, however, that in view of the extensive gauze drainage the probabilities were that obstruction had been produced, and I did not consider it wise to delay for more definite symptoms.

Second operation for obstruction, twenty-seven hours after drainage of abscess, November 15th, 3 P.M. The closed incision above the gauze drainage in the middle line was opened; the packing apparently had remained in place undisturbed. It was surrounded on the upper and left side by coils of slightly adherent and considerably distended small intestine, and to the right side by the cæcum, which was not distended; to the lower side by the bladder. The transverse colon, which was exposed, was not distended. On separating the coils of small intestine from the gauze packing there was an exudate of fibrin and cloudy fluid, indicating at least beginning local peritonitis. The abdominal cavity about the gauze drainage was protected by new strips of gauze and then the packing of the abscess was removed. There had been some little leakage, and the cavity had contracted down to the diameter of the gauze introduced into the abscess cavity, about 5 x 3 cm. The cavity was lined, not by granulation tissue, but by a slightly hemorrhagic, necrotic exudate, and I found two or three round worms. This cavity was sponged out and again filled with bismuth gauze. A second incision was made to the right of the cæcum, and a piece of gauze was passed through the mesentery of the cæcum into the abscess cavity. I

think now that this was an unnecessary procedure, but I felt at that time that drainage from this side might allow an earlier removal of the drainage into the cavity from the middle line; but one could only make a small opening through the mesentery of the cæcum, as a larger opening would have jeopardized the blood supply. This completed the toilet for the drainage of the abscess. About the gauze which extended into the abscess cavity more gauze was placed in order to protect the general peritoneal cavity. I then selected a loop of small intestine as near as I could to the cæcum, but a loop that would not come in contact with the gauze drainage. I felt that if I selected a loop in contact with the gauze drainage it might become obstructed. This loop was brought out of the middle-line incision above the umbilicus and held in place by gauze packing between the loop and the parietal peritoneum. Before suturing this loop in place it was opened and the distended gut emptied of its fluid and gaseous contents. The large accumulation of gas and fluid in the upper loops of the small intestine and the non-distended condition of the small intestine near the cæcum were pretty clear evidences of obstruction. I considered that if I did not empty the upper portions of the small intestine during the operation I could not expect with certainty that they would empty themselves. In addition, the immediate relief of the marked distention gave more room for proper drainage and partial closure of the wound. The condition of the wound at the end of the operation was as follows: In the upper end of the incision the open loop of the intestine surrounded by gauze; the next 3 cm. the recti muscles were approximated with silver wire; below this the gauze packing protecting the general peritoneal cavity; then the gauze and rubber protective drainage into the abscess cavity.

Recovery from the Second Operation. The patient was considerably shocked. The temperature arose quickly to 103.6° F., pulse 160, respiration 28, leucocyte count 25,000. Six hours after operation a subcutaneous infusion of salt solution was given. Three hours later the patient was critically ill. Bacilli were found in the blood. The clinical picture was one of acute toxæmia. At this time Dr. Hume gave the patient an intravenous infusion of 325 c.c. of silver nitrate solution. Within twelve hours her condition was very much better. In forty-eight hours the patient had completely recovered from the shock.

During the remainder of the child's illness there were no complications to be dealt with except the eczema produced by the fecal discharge and the condition of starvation.

The Treatment of the Fecal Fistula. From November 15th to December 15th the fecal fistula demanded almost hourly attention. For the first few days we were able to collect the fecal matter from the upper loop of the intestine through a rubber drainage-tube, but about this time it became almost impossible to keep the tube in place. Three or four round worms perforated the wall of the intestine about the tube and weakened the wall. The opening became larger and allowed leakage around the tube; any attempt at high introduction of the tube met with some obstruction. The leakage produced an extensive eczema of the skin of the abdomen. The patient did not stand the continuous bath treatment, which was tried for two or three days. A number of methods were attempted to protect the skin from the fecal discharge. Finally, Dr. Fisher succeeded in placing

a piece of rubber dam used by dentists about the intestinal opening in such a manner that the contents of the intestine passed through an opening in the rubber were collected there. In this way the skin was protected. Our usual method in fecal fistula is to keep a rubber drainage-tube in the upper loop to collect the discharge and every few hours to pass a catheter into the lower loop, and then introduce the contents collected from the upper portion of the intestine into the lower; that is, to feed the patient into the lower portion of the intestine with the partly digested contents of the upper. I used this method first in 1897 in a case of strangulated femoral hernia.[1]

Unfortunately this method could not be used in this case. A permanent tube could not be introduced far enough into the upper loop of the intestine to prevent leakage, and the obstruction prevented feeding in the lower loop. This obstruction was not relieved until about December 13th, almost four weeks after the operation. At this time we were able to demonstrate that methylene blue introduced into the lower small intestine appeared in the discharge from the rectum, and also after feeding through the lower loop normal stools were ejected from the rectum. Having demonstrated that the obstruction was relieved, the condition of the patient demanded that the fecal fistula be closed.

Closure of Fecal Fistula. On December 15th, the twenty-ninth day, without anæsthesia, the loop of the intestine in the upper portion of the wound which had been opened was partially separated, drawn partly out of the wound, and a rather rough lateral anastomosis made just below the divided ends. These were then closed. The suture was difficult because the peritoneal surfaces were covered with granulation tissue. I felt that an attempt of this kind should be made first, because the general condition of the child contraindicated anæsthesia, and the condition of the skin, although it had improved very much since the use of the rubber dam, was still sufficiently bad, and it would have been difficult to prevent infection if the general peritoneal cavity had been opened. Fortunately, this suture held perfectly for three days. During this time the eczema practically disappeared and the child's strength increased; there was no question that the obstruction had been relieved, as there were normal stools. During the next four days the suture began to leak and demanded daily patching. For this reason on December 22d I felt that the condition of the child and the skin would allow an opening of the peritoneal cavity without great danger. This was done to the left of the rectus, and two loops of intestine adherent to the wound which appeared to be the upper and lower loop were anastomosed by lateral suture. I did not think it was justifiable to separate all the adhesions and so make certain of the proper loops of the intestine. Fearing, however, that the anastomosis might have been made between an upper or only a lower portion of the intestine, I patched up the original suture of the gut in the wound. Following this operation it was quickly demonstrated that the lateral suture had failed to restore the continuity of the alimentary canal. The patched-up suture in the wound was soon beyond further repair. Fortunately, however, these two temporary sutures in the wound had allowed the skin to completely recover from its eczema, and the restoration of the alimen-

[1] Operations on 459 Cases of Hernia, etc., Johns Hopkins Hospital Reports, vol. vii.

tary canal to its complete function gave the patient opportunity to gain in strength. The moment the temporary suture became irreparable, on December 27th, I decided to make the attempt at a permanent anastomosis of the two loops in the wound. The temporary suture had performed good service for almost eleven days. Fortunately, at this time, all the original gauze drainage into the abscess cavity and its surroundings had been removed, and the wound was practically closed up to the fecal fistula. At this last operation on December 27th the two portions of the intestine in the wound were separated and drawn out of the peritoneal cavity until there was at least 6 cm. of normal peritoneal surface. I was surprised at the few adhesions. They extended hardly more than 1 cm. beyond the parietal peritoneum of the wound. There was little difficulty in separating and withdrawing these two loops of intestine and at the same time protecting the peritoneal cavity with gauze. That portion of these two loops forming the fecal fistula which was covered with granulation tissue was excised. This left practically normal intestine. A long lateral anastomosis was then made—at least 3½ cm.—beginning about 1½ cm. from the cut end. The ends were invaginated and closed. The anastomosed gut was allowed to drop back into the peritoneal cavity. It, however, was not pushed back. About 1½ cm. projected beyond the skin. It was left in this position and surrounded with a few pieces of gauze. Only two sutures were placed in the lower portion of the wound. I considered that a lateral suture was much safer than an end-to-end, and that it was better not to make any attempt at complete closure of the wound. The result apparently has justified this procedure. The suture held. The projecting anastomosed loops were rapidly withdrawn into the abdominal cavity and, after the removal of the gauze, became adherent to the recti muscles, and within three weeks the wound closed over with granulation tissue, and to-night, four weeks after operation, is completely covered with epidermis. The child has never exhibited symptoms of obstruction since the anastomosis.

In reporting the fortunate result of such a difficult and complicated surgical case it is only just to add that such results in surgery would be impossible except in a well-equipped hospital. In this instance especially I am convinced that the operative surgery would have been of little avail without the faithful and skilled attention of the trained nurse and the constant vigilance of the resident surgeon.

THE RESULTS IN BLOODLESS REPOSITION OF CONGENITAL DISLOCATION OF THE HIPS, WITH A REPORT OF CASES.

By Gwilym G. Davis, M.D.,
of Philadelphia.

The cases reported were all subjected to forcible correction two to five years ago.

Case I.—Boy, aged three years; walked since the age of thirteen months. There was marked lordosis, limping, and a shortening of the affected (right) limb of one and a quarter inches (3.25 cm.). He

was placed in bed and extension of four pounds applied, with the limb in an abducted position, for eight weeks. He was then etherized (December 1, 1900), the adductor muscles cut, and the head of the femur replaced by flexion, abduction, and outward rotation, as advised by Paci. As reluxation occurred on attempting complete extension of the limb, it was bound in an abducted and externally rotated position on a straight posterior splint, which extended from beneath the back out along the posterior surface of the thigh to the knee, and he was confined to bed. The luxation recurring, the bone was again replaced and extension of four pounds, with the limb in the abducted position, was again applied. At the end of two months the leg was rotated strongly outward and the limb placed in plaster in marked abduction. At this time the head readily left its new position. Two months later the plaster was changed, the limb brought somewhat down, and a new plaster cast applied, with the limb markedly everted. The head now seemed fixed, and a few weeks later he was allowed to walk around. The plaster cast was afterward replaced with a metal brace, which held the foot in marked eversion.

On examining him now, two years and five months after reposition, we find about 2 cm. (four-fifths inch) shortening—a gain of 1.25 cm. The lordosis has almost entirely disappeared. The movement up and down in the hip is no longer present, and the head is now firmly fixed at the upper anterior edge of the acetabular region. It is what is termed an upward and anterior transposition. Instead of the head being loose on the dorsum of the ilium above and posterior to the acetabulum, it is now firmly fixed at its upper anterior edge. The functional result is good, as the movements of the hip are free and painless; he uses roller-skates, but there is still some limping, which is not at all marked. This is not an example of a perfect result, but it is a satisfactory result, as the only disability which remains is that due to shortening, which can be compensated by the high shoe. There is twisting of the neck in this case.

Case II.—A girl, aged four years, born after a difficult parturition. Had been walking for a year and seven months previous to applying for treatment. Her peculiar waddling gait was noticed since she began walking. She was very unstable on her feet, falling readily. Lordosis was marked, the left trochanter being 3.5 cm. and the right 3.75 cm. (one and a half inches) above Nélaton's line. She was put to bed for six weeks, with first three and then five pounds' extension, with the limbs widely extended. She was then etherized, the adductor longus tendons cut, and both hips were readily reduced by the manipulations used in the previous case. A plaster cast was then applied, holding the limbs abducted not quite to a right angle, and rotated out so that the heels and toes were in the same transverse straight line. For convenience in handling and strength, a splint was fastened across the thighs from one to the other just above the knees. After two months a new cast was applied and the limbs brought partly down. As the right hip showed a tendency to slip out of position, a new cast was applied and continued below the knees. Seven months after reposition the right leg swelled and showed evidences of pressure (probably by the anteverted head pressing the femoral vein against the cast), so the cast was removed and reapplied. Two months later a plaster cast was applied to the hips and thighs and continued down

with jointed side irons to the shoes, where they were fastened, keeping the feet everted. She now began to walk a little sideways by holding onto the bed. One month later, ten months after reposition, she walked with crutches. All apparatus has been discontinued for more than a year.

On examining her, now two years and five months after operation, the left hip is found to be apparently normal in every respect. The right hip is found to be somewhat higher up and anterior than normal. On measuring it is found to be 2 cm. (four-fifths inch) shorter than the left. In other words, it is in a position of anterior and upward transposition. It seems firm and does not slide up and down when pressure is made on it. The lordosis is gone, but she still limps, which may partly be removed by the elevation of the sole of the shoe.

CASE III. was a girl, aged three years, with both hips dorsally luxated. The trochanters were very prominent and she walked with difficulty, wabbling from side to side.

She was etherized, the adductors cut, and both hips replaced by manipulation. She was put up in plaster, the legs being abducted in the frog position, not quite at right angles to the body. In five weeks another cast was applied and left for another month. One hip was then found to be out. The cast was reapplied and a month later the hip was again found to be out. Operation was advised and refused. An extension brace was then applied and the child allowed to go around on crutches. After coming for awhile as an out-patient the case disappeared, the father being apparently desirous of escaping paying for the apparatus, and it was impossible to find him.

In this case one hip remained in place ever since its first replacement and never showed any tendency to come out again, even though the child had been walking on it for a couple of months. This case was lost sight of about six months after replacement. A fair result could have been obtained by brace treatment in the other hip also had attendance been continued.

CASE IV.—A girl, aged eight years, I showed to the Pediatric Society over three years ago. One hip was replaced five years ago by manipulation, the other by operation. A skiagram demonstrates a perfect anatomical result in the hip replaced by manipulation, and that the functional result in this hip is absolutely perfect now, five years later. The operated hip is firmly fixed in an anterior upward transposition.

To sum up, out of seven dislocated hips in four patients, one was operated on and one was lost sight of while under treatment. Of the other five, two were anterior transpositions and three were apparently perfectly normal in every way five years, two and a half years, and the last six months after replacement by manipulation.

Of the two anterior transpositions, one was a single dislocation in a boy, with a twisting of the neck of the femur; and while there is some shortening and limp remaining after two years and five months, the result is still satisfactory, because the head has become fixed and the centre of gravity has been thrown farther posteriorly. The former prevents any further increase of disability as age advances; on the contrary, the functional results will probably improve rather than

retrograde. The latter removes the tendency to lordosis, which later in life may prove very distressing.

The other transposition occurred in Case II., a double one, in which the other hip remained reduced in the acetabulum. This leg showed a shortening of about four-fifths of an inch, and the resulting limp is quite distinct. The fact, however, that one has been able to reduce one hip perfectly and fix the other one makes the result in this case also a satisfactory one, as the lordosis and tendency to increase have both been eliminated and the walking much improved. She plays and goes to school without getting tired. Previous to treatment the disability in this child was quite marked, as she was very unstable on her legs and frequently fell down. It is now two years and five months since reposition.

As regards the method of reduction, it is too intricate a subject to be discussed at this time; but I might state that these hips were practically all reduced by the method of circumduction as practised in traumatic luxations and as applied to congenital and pathological luxations by Paci, of Pisa, since 1888, with the addition of tenotomy of the adductor longus tendon.

There are two principal ways of replacing congenitally luxated hips. One is by an open operation in which the way for the head is cleared by the knife, and the other is forcible reposition, combined or not with extension and tenotomy. The latter has also been called the bloodless method in contradistinction to the open cutting operation.

Let us inquire into the results of the method by forcible reposition. In so doing it is essential that we bear in mind that different operators have different standards of what constitutes a cure. An exact reporter will of necessity have less so-called cures than an inexact one, and yet the results of the former may be better than those of the latter. The difference in the results of various cases is comparatively slight. They shade gradually from the bad into the good, and it is very difficult to draw a positive line of distinction. The age also has a marked influence on the results, and the older the patient the more difficult is it to achieve good results. There is likewise a considerable difference in the cases themselves, and this modifies the results. It is obvious that, with deformed femoral heads and necks and undeveloped acetabula, perfect results are an impossibility.

The difficulty of securing absolutely perfect results caused Paci to be extremely conservative as regards what was achieved. He only professed to put the head as near as possible in the acetabulum, and in his earlier writings, at least, he did not claim to put the head absolutely back into a normal position. It was only after he had been working six or seven years that, in 1894, he was able to obtain a specimen post-mortem which had been replaced by his method by A. Nota,

of Turin, four months previously. This showed both acetabula well formed, but one better than the other, thus conclusively demonstrating what had been doubted previously by Lovett, Lorenz, Kirmisson, and others. Thus we may accept as a demonstrated fact that within a period of four months from the time of reposition it is possible for at least a certain proportion of the replaced hips to have again resumed a practically normal condition. Kirmisson has always held that true repositions were very rare, and the conservative position of Paci has already been alluded to. When Lorenz, however, began practising the forcible reposition, he claimed that his results were absolutely curative and that the reposition was exact.

Kirmisson replied to this at the Paris International Congress in 1900, and showed that the skiagraph has demonstrated his former position to be correct; and Lorenz himself now speaks of "anatomical results," by which is meant that the head of the bone is shown apparently well seated in a socket, with a good overhanging edge to support it. Cases in which a displacement of the head can be shown are spoken of as transpositions. In these the head is usually fixed either anteriorly or anteriorly and upward. From this it is readily seen that when writers speak of a cure, one is not certain what is the actual condition obtained. Even when the result is a so-called cure by transposition, the functional results may be and usually are excellent. What some operators would call anatomical results (or cures) others might call transpositions.

When one considers the immediate results they may be either good or bad. If good, the head may be replaced at least approximately at the site of the original acetabulum with no serious effects from the manipulations employed. If bad, there may be a failure to replace the bone and serious injuries may be occasioned by the efforts at reduction. That the danger of serious injury being produced was appreciated by Paci is evidenced by his carefully cautioning against the use of undue force, for fear of fracturing the femur. The manner in which Lorenz applies forcible reduction is, however, mainly characterized by the exercise of great force; therefore, one is not surprised to find the resultant injuries both numercus and extensive. Paci in some of his cases used such mild manipulations as not to require an anæsthetic. Lorenz[1] states that in 450 cases he had three deaths from chloroform and shock alone; in addition there were eleven fractures of the neck of the femur, one of the pubic bone, one of the ilium, three paralyses of the peroneal nerve, seven of the anterior crural, besides some of the sciatic, and one total gangrene of the extremity. There were also instances of stiffness of the joint and the serious ruptures known as Narath herniæ.

[1] Heilung der angeborenen Hüftgelenks-Verrenkung, Wien, 1900.

Heusner, of Barmen,[1] says that he and Hoffa have had even more serious results. Hoffa (German Congress, 1899) has had tearing of the soft parts (vulva and urethra), fractures and separation of the epiphyses, paralysis of the sciatic and anterior crural nerves, and suppuration of the hæmatomas which formed at the site of the rupture of the adductors. He even lost one child of six years.

Drs. Wilson and Dinkelspiel state that one of the patients operated on by Prof. Lorenz in this city during his recent visit was also seized with convulsions and went into a stupor, which, however, did not end fatally.

The dangers of circulatory disturbances occurring is shown by my Case II., in which some time after reposition the leg became swollen and blue and the cast had to be removed. This was due to the head, as it came forward, pressing the femoral vein against the inside of the cast. The Narath herniæ alluded to were described by Narath,[2] of Utrecht, who said they had occurred in 12 per cent. of his cases. They were large herniæ which came down beneath Poupart's ligament, and were supposed to be due to tearing of the fascia lata and other tissues in front of the head of the femur. Lorenz, both by his writings and demonstrations, has advocated the exercise of such extreme force in attempting reductions that his followers will probably repeat some of his bad as well as his good results.

When we come to consider the ultimate results we find that we must allow some time to elapse before assuming them to be definite. When a congenitally displaced hip has been replaced it is not as if the case was one of traumatic luxation. In the latter a normal head goes back to a normal acetabulum and stays there; in the congenital a more or less altered head goes back to a more or less altered acetabulum and often does not stay there. In Case IV., one of congenital double luxation, on one side the hip remained in place, but the other persistently luxated and was, therefore, operated on by open incision. One hip in Case III. persistently reluxated and was put under treatment by apparatus. One hip of Case II. also came out, but was eventually fixed in an anterior transposition. In Case I., of single luxation, the head was only fixed by repeated reluxations.

This tendency to reluxation seems to exist in approximately one-half the cases. In my own experience the cases that have remained reduced have done well from the start, and have shown but little tendency to reluxation at any time after replacement. The exact position of the head of the femur cannot be determined while the fixation bandage is on. Therefore, it is at the time of the first removal of the plaster

[1] Zeitschrift für orthopedische Chirurgie, 1902.
[2] Report of German Surgical Congress, 1899.

bandage that one is able to determine whether or not reduction has been maintained. If it has not, reposition must again be performed. In most of the cases which show a more or less marked tendency to reluxation a good result can be obtained by means of prolonged treatment. They usually constitute the cases known as transpositions, in which the head of the femur, instead of being placed exactly in the acetabulum, is more toward its upper edge or upper anterior edge, near the anterior inferior spine of the ilium. These patients can usually walk fairly well without marked fatigue. The limb is usually shorter than normal, but its range of movement is good. Some limping usually persists in spite of a high shoe, and if the case is a double one, with one hip replaced and the other transposed, the limping may be marked. The lordosis is in most cases nearly or quite abolished. It is evident that good results cannot be expected if the bones are abnormal. The neck or shaft of the femur may be twisted so that when the foot points forward the head is in a position of anterior transposition. Some of these cases can be remedied by doing a subtrochanteric osteotomy and turning the foot more outward.

In other cases instead of the head and neck forming an angle of 130 degrees or more with the shaft it may form a right angle or less. This causes a condition known as coxa vara, and the thigh is more or less fixed in an adducted position. Abduction in these cases is much restricted. Shortening is also present. When there is a deformity or partial lack of the cotyloid cavity and head of the femur, complete failure might occur, or considerable shortening with a more or less marked coxa vara condition, even if one was successful in fixing the head.

Let us now look at some of the statistics which have been brought forward as illustrating the results of the method of forcible reposition:

Lorenz, in his book published in 1900, gives, out of 212 cases, 108 anatomical cures in which the skiagraph showed the head under a good, overhanging, acetabular edge. Of these 108, 61 were one-sided luxations occurring in 102 cases, and 47 were in children with double luxations and occurred in 110 joints in 55 children. Subluxation occurred in 20 cases and forward and upward luxation in 82 cases.

Hoffa,[1] out of 64 cases, 42 single and 22 double, reduced all; they varied from two to eight years in age. Great injuries were done to the soft parts, etc. Of the 42 single repositions, 4 remained cured and 1 of these was found to have a condition of coxa vara. In 1 case complete reluxation followed; in 25 the head was transposed in front and up, and in 11 more anterior, near the anterior inferior spine. Of the 22 double luxations, in 3 both sides reluxated; in 4 there was

[1] Archiv für klin. Chirurg., Bd. lix., S. 525.

reposition on one side and transposition on the other, and in the remaining 15 there was transposition in the hips of both sides.

Max Schede's clinic, as given by Petersen,[1] in 113 cases, 61 single and 52 double, there were 6 cures; in 48 the head was near the correct position in the acetabulum, and in 59 it was transposed anteriorly.

Mikulicz's clinic, as given by Drehmann,[2] in 131 cases there was failure to replace in 7, 1 of which was over ten years of age and the other 6 from four to ten years. In 118 repositions, operation was later done in 6 for reluxations; in 4 cases the results were not known, and 2 cases died, leaving 106—82 single, 42 double; 87 of these have been under treatment one-half year or more—66 single, 21 double. Of the 66 single, in 48 reposition remained and in 18 was transposed.

Of the 21 double cases, with 42 hips, 6 failed, 14 were transposed, and 22 remained in position.

Heusner (German Congress, 1899) considered in 10 per cent. the results excellent, in 30 to 50 per cent. good after long treatment, and in the remainder unpromising.

Karl Ludloff, in 11 cases, claims satisfactory results in 9; 2 were nearly ideal, 4 were good, 3 showed coxa vara; in 1 was a partial reluxation, and in 1 there was a double coxa vara with marked restriction of motion.

Among others, Kollicker, in 25 single and 13 double cases, claimed only two cures; Codivilla, in 66 cases, four to five, and Kümmel about 5 per cent., stating that unfortuate occurrences are not rare.

The experiences of American surgeons—Bradford, Whitman, Sherman, Ridlon, and others—have been much like those already related.

In considering the various facts here presented, one may fairly conclude that Kirmisson had good grounds for stating that perfect reposition was rarely attained. They also furnish evidence of the good judgment of Paci in refraining from claiming for his method an absolute cure. If one is critical, as is apparently the case with Kirmisson, Hoffa, Kollicker, Schede, Codivilla, and others, then one will call 5 per cent. cured. If one is more easily satisfied, then the cures will amount to 50 per cent., as in the case of Lorenz, or more, as in the case of Mikulicz. The truth probably is that the results in the hands of the various operators do not differ so much as do their statistics. It appears to be the fact that total failures below the age of six to eight years are comparatively rare; that approximately one-half the cases are replaced in a more or less normal acetabulum and are kept there; that a small proportion of these are apparently quite normal; that in most of the remainder the head is transposed forward or forward and upward and

[1] Deutsche Zeitschrift für Chirurgie, Bd. l., S. 534.
[2] Zeitschrift für Orthopædische Chirurgie, 1903, p. 304.

remains in this position. In many of these cases of partial repositions the condition can be subsequently improved, as pointed out by Whitman, Max Schede, Bradford, Kirmisson, and others, by subsequent operations, as osteotomy. A certain number of cases seem to do well from the moment of reposition and remain in place without trouble; perhaps half show more or less of a tendency either to slip backward into their previous luxated position or anterior. These are to be treated by subsequent reposition and reapplication of the bandages and the use of various forms of apparatus. To speak of a cure soon after reposition has been performed is ridiculous. Lorenz stated that he did not bring the legs parallel to each other for two years after reposition. The exact power possessed by the human economy to form a new joint is as yet a matter of doubt. The pathological preparations obtained of joints which had been replaced some time before death, as those of Paci, Bradford, Ochsner, and others, show that good joints do form, but they may have been nearly normal at the time of reposition. The fact that deformities of the head, neck, and acetabulum may exist should cause one to be guarded in promising as to the results to be achieved by treatment in any given case.

TORTUOSITY OF THE AORTA.

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AND

G. E. Pfahler, M.D.,

OF PHILADELPHIA.

In the course of a series of studies upon lesions of the aorta, which consisted of careful clinical examinations combined with fluoroscopic and radiographic examinations, we were surprised at the frequency with which some of the lesser signs of aortic aneurism were present. Among these were inequality of the radial pulses, suprasternal pulsation, tracheal tugging, accentuation of the aortic second sound, and brassy cough. Not infrequently the patients complained of certain other symptoms, particularly dyspnœa, anginoid attacks, and localized pains. In many cases aneurism was undoubtedly present, either causing characteristic pressure symptoms or being manifest as an external expansile pulsation. In a number of cases, however, we observed with the fluoroscope an expansion in the shadow of the aorta at the level of the fifth or sixth dorsal vertebra, extending from one-half to two inches to the left. This, we supposed, represented an aneurismal dilatation of the aorta at this point. Upon all cases that died effort was made to obtain autop-

sies, sometimes successfully; and we discovered that in the majority of cases in which this skiagraphic picture was observed no dilatation of the aorta was present, nor did sections through the wall of the aorta at this point indicate that there was any diminution of the muscular layer, such as might have accounted for dilatation under pressure of the blood

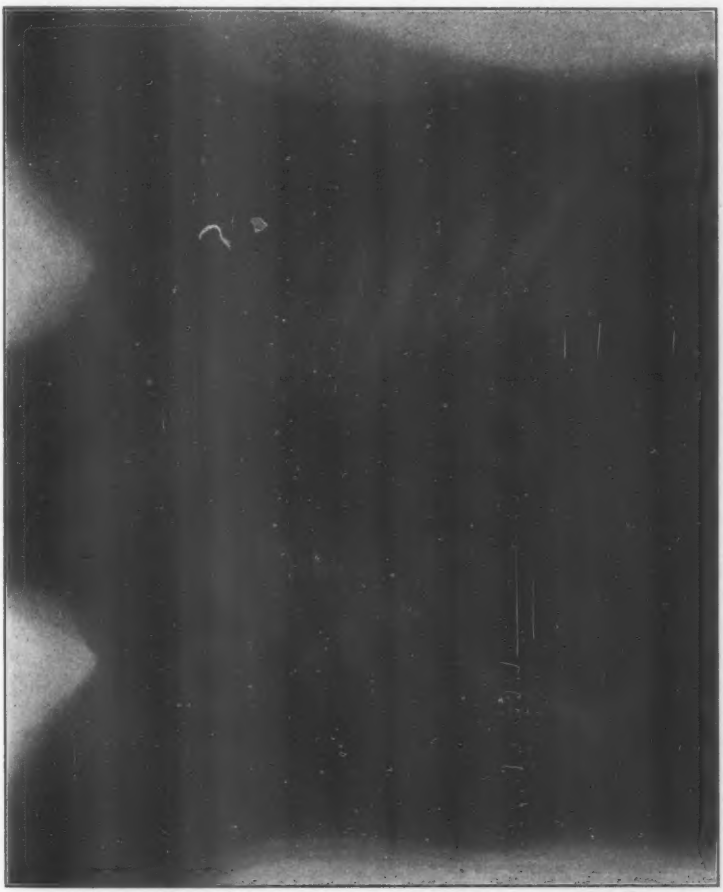

FIG. 1.

Normal chest. No bulging in the shadow of the aorta at the junction of the transverse and descending portion.

current. In the second autopsy careful dissection showed that at this point the aorta made a bend to the left, not very unlike the tortuosity that is observed frequently in cases of atheroma of the peripheral arteries. Altogether we have records of eighteen cases with skiagraphs in which this combination occurred, and of these four have been exam-

ined post-mortem, all with similar results. It seems not unlikely, therefore, that among subjects of arterial sclerosis this tortuosity of the aorta, giving rise to some of the signs of aneurism of the arch, is relatively a common process. There is no reason to suppose that tortuosity of the arch of the aorta differs in any way from tortuosity of the other

FIG. 2.

Tortuosity of the aorta. The dark shadow of the spinal column may be seen in the central portion of the skiagraph. The shadow of the aorta extends upward about one inch to the right of the spinal column, and at the left makes a sharp bend about two inches to the left, then curves inward and downward, where it joins the shadow of the heart.

arteries of the body. In all probability it is produced by a loss of elasticity, a gradual elongation and dilatation of the bloodvessel as a result of this loss, and the fact that at certain points in its course the artery is fixed, so that between these points compensation for elongation is only accomplished by bending. It would seem as if in the aorta

this compensation could be attained by increase in the height of the arch. The arch of the aorta is practically fixed at two points: the aortic orifice of the left ventricle, and the point where it reaches the

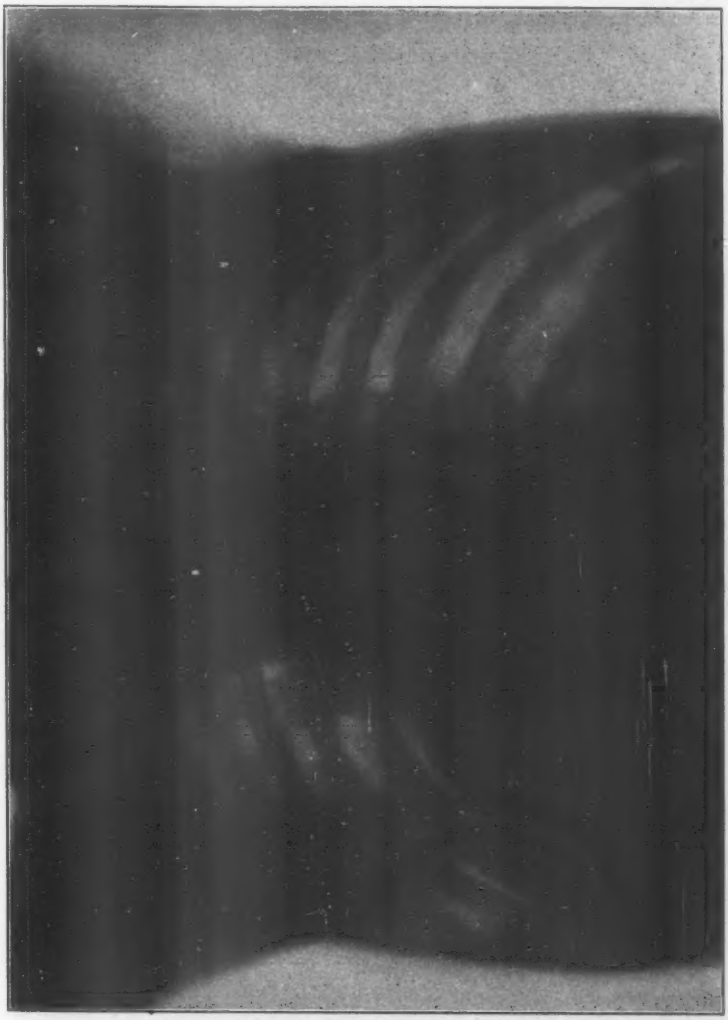

FIG. 3.

Tortuosity of the aorta, shown by the projection in the shadow at the left portion of the arch.

vertebral column at the level of the fifth dorsal vertebra. The position of the attachment to the heart can only vary when the heart itself is moved; the attachment to the vertebra is not quite so firm and is made

by connective tissue and by the intercostal arteries. Nevertheless, it is sufficiently firm to prevent extension downward, in which it is aided by the large vessels arising from the arch. The extension upward of

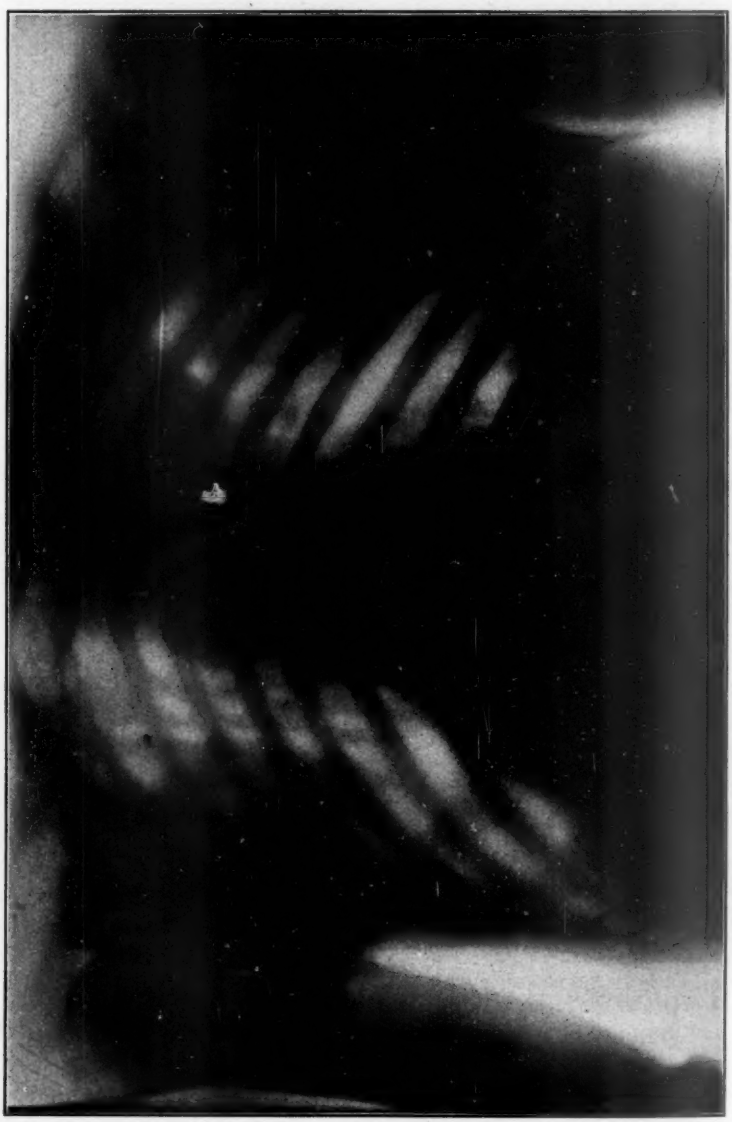

FIG. 4.

Tortuosity of the aorta and aneurism of the innominate artery. The shadow of the aorta may be seen extending upward to the right of the spinal column, where it is continuous with the shadow of the innominate aneurism; then a bulging is seen at the left portion of the arch, which is indicative of the tortuosity.

FIG. 5.

the arch is hindered by the closeness of the manubrium to the bodies of the vertebra and the fact that the space between them contains the trachea, the œsophagus, and the vessels arising from the arch. That a certain amount of extension upward usually does occur is evidenced by

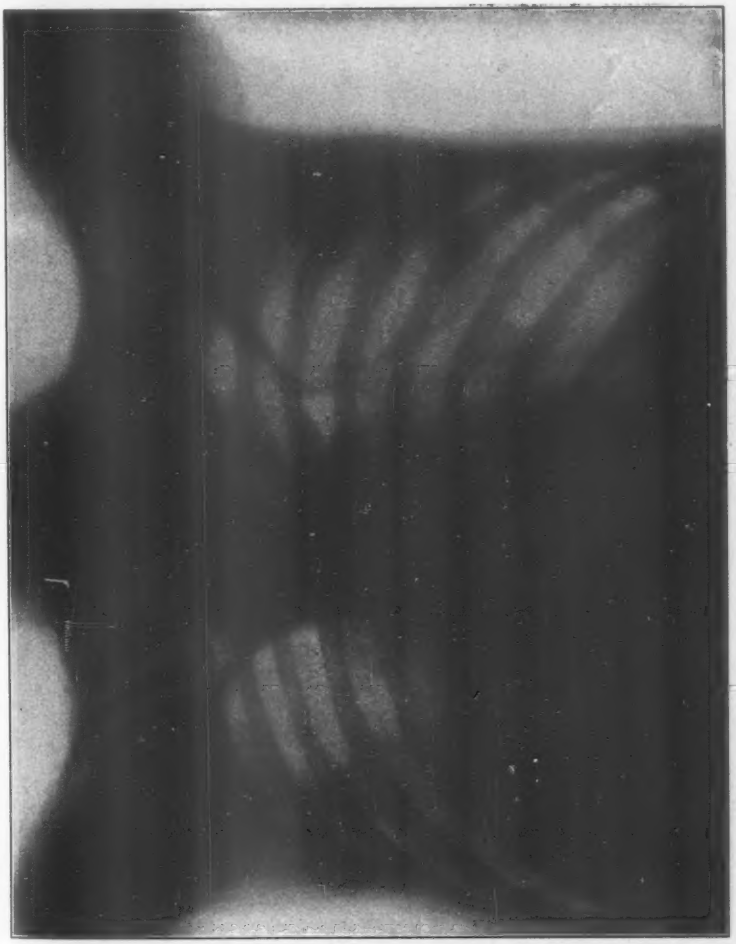

FIG. 5.

Tortuosity of the aorta, shown by the projection in the shadow at the left portion of the arch.

the frequency of suprasternal pulsation, but this extension can only be moderate, and the elongation of the aorta, exposed as it is to the full force of the contraction of the left ventricle, in all probability must be frequently so great that it cannot be compensated entirely by this

movement; therefore it is obliged to deviate laterally, in which direction it meets little to resist it except the compressible upper lobe of the left lung. As a result of this, the descending portion, before it reaches the vertebral column, becomes separated further than normally from it,

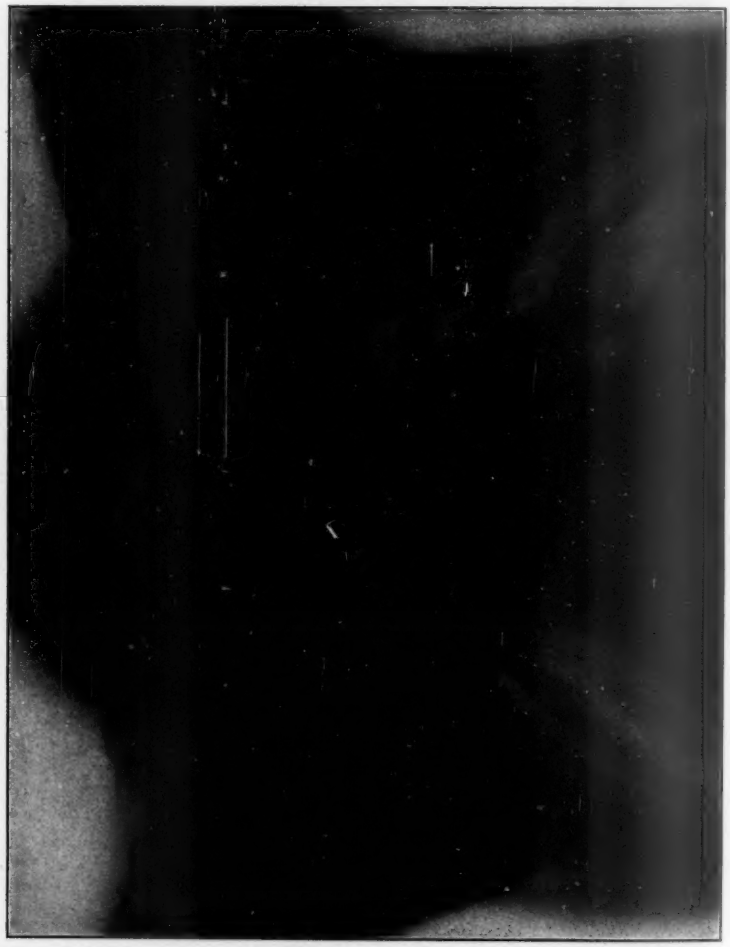

FIG. 6.

Tortuosity of the aorta. This is shown by the marked bulging of the shadow at the junction of the transverse and descending portions of the arch of the aorta.

so that it produces a slight bending at the point where they join, and this bend is the tortuosity that we have observed in our skiagrams. The examinations of the aorta in old persons have convinced us that this tortuosity is by no means uncommon, even in patients who have

not necessarily had clinical symptoms that attracted attention. If at an autopsy or a dissection, after the removal of the sternum, the left lung is carefully dissected away without disturbing the position of the

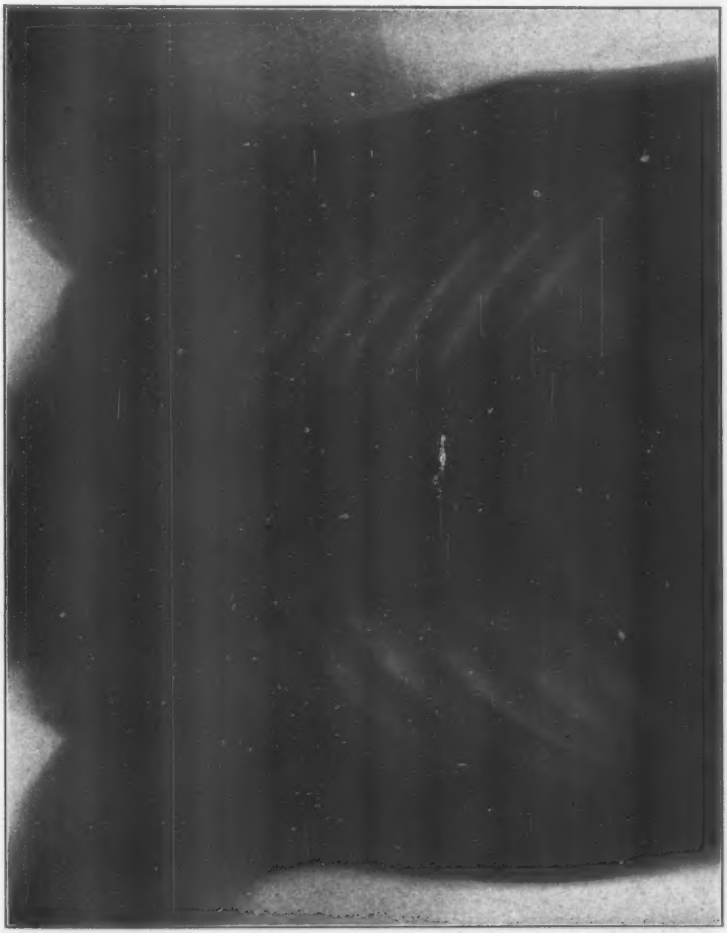

FIG. 7.

Tortuosity of the aorta. This is indicated by an increase of about one inch in the shadow of the aorta at the junction of the transverse and descending portions.

heart, aorta, or right lung, the whole arch of the aorta is then exposed and any irregularity readily demonstrated.

Atheroma of the aorta has received considerable attention from clinicians. The physical signs that it is supposed to cause, however, have been, as a rule, limited to an impairment of the resonant note over the

upper portion of the sternum, which may extend slightly to either side of its border, and is due to dilatation of the first portion of the aorta; a systolic murmur, heard best over the manubrium and transmitted into

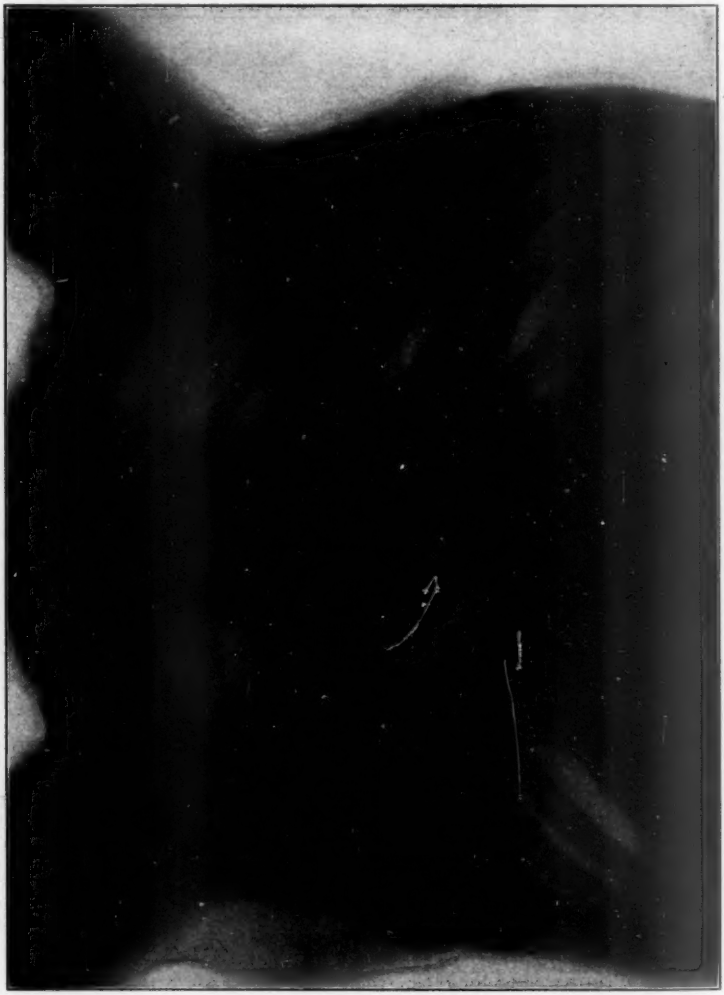

FIG. 6.

Tortuosity of the aorta, indicated by the bulging of the shadow at the left portion of the arch.

the vessels of the neck, and occasionally—it is stated—heard also at the apex; and sometimes a double murmur, due less to the atheromatous condition than to the insufficiency of the aortic valves, which is secondary

to it. Sansom[1] mentions the fact that sometimes the arch of the aorta is elevated, causing elevation of the left subclavian and the innominate, and rendering these arteries more susceptible to palpation, so that it is easy to produce a thrill in them on pressure; and occasionally the thrill occurs spontaneously. The symptoms of atheroma are pain, dyspnœa, and sometimes anginoid attacks. We have been unable to find any reference to inequality of the pulses, tracheal tugging, inequality of the pupils, and suprasternal pulsation. There is some difference of opinion as to whether the second aortic sound is invariably accentuated or not. It does not appear to have been recorded that typical aneurismal cough may occur as a result of an atheromatous condition without aneurismal dilatation.

The investigators, who employed the fluoroscope or skiagrams for the purpose of studying changes in the aorta, were early impressed by the fact that although aneurism was occasionally overlooked, it was more frequently diagnosed when it did not exist. In 1897 Levy-Dorne[2] stated that the early stages of aneurism were more common than had hitherto been supposed, and that the prognosis of commencing aneurism appeared to be not so unfavorable, especially as only a few had any tendency to enlarge. His pictures illustrate the small pulsating projections to the left of the sternum. Gocht,[3] a year later, thought that by means of the Röntgen ray it was possible for the clinician to determine the presence of aneurism in cases in which doubtful symptoms were present. In opposition to this, Dumstrey and Metzner[4] recommend considerable caution in the diagnosis of aneurism by means of the Röntgen rays, especially when there are no physical symptoms. They thought that mediastinal tumors might give rise to the same picture. Holzknecht[5] was the first to show that pulsating hemispherical shadows just above the heart are not always aneurisms. He argued that this idea was based upon the supposition that the thoracic aorta was always situated in the median line, and that the arch lies in the shadow of the sternum. This is true only of the ascending portion of the arch, and as the transverse and descending portions lie practically in the same plane, they are projected as a pulsating, band-like shadow, intensely dark and with a free end. It may be seen during life, although he agrees with Weinberger[6] that it rarely projects considerably to the left. This projection has also been observed and described by Immelmann.[7] Holzknecht later discussed the subject more thoroughly in his text-book. In this he stated that aneurism of the arch theoretically was usually seen to the right of the sternum in sagittal pictures. Dilatation gives rise to a shadow seen on either side of the sternum, and is readily confused with aneurism. The differential diagnosis can be made by the fact that there are no pressure symptoms and no tendency of the shadow to enlarge. Simple dislocation of the aorta usually appears

just to the left of the spinal column, and the pulsating shadow sometimes extends as much as six inches to the left of the left border of the sternum, forming, in fact, a pulsating shadow much larger than that projected by aneurism of the arch. In cases in which the heart is elevated the natural bending of the heart to the left is increased. In forty-six cases that he observed in which this shadow was present only six proved ultimately to be cases of aneurism. Holzknecht has undoubtedly seen and correctly interpreted the picture to which we call attention; but he classifies all these conditions as dislocation of the aorta, and fails to recognize that there is usually an actual elongation of the vessel, and that, in addition to the thrusting of the arch to one side, there is also a condition of tortuosity.

The following are the abridged notes of the cases that we observed:

CASE I.—P. H., aged thirty-eight years, a negro hostler. Had syphilis at the age of fifteen years. Eighteen months before death he received a blow over the right clavicle, and later noticed pulsation in the right side of the neck, followed by pain, cough, and paroxysms of dyspnœa.

Physical Examination. The pupils were equal. The right radial pulse was absent; the left radial pulse was small. The right carotid pulse was absent, and the left carotid pulse and its branches pulsated vigorously. There was pulsation of the prominent sternal end of the right clavicle, due to a mass beneath in which there was neither thrill nor murmur. The right external jugular vein was distended. The area of cardiac dulness was normal. The aortic second sound was accentuated. A diagnosis of innominate aneurism was made, but it was difficult to explain why the left radial pulse was so weak. The fluoroscope showed a pulsating tumor in the region of the innominate, and a prominence to the left of the descending aorta that had expansile pulsation. At the autopsy there was found aneurism of the innominate artery and atheroma and tortuosity of the arch of the aorta.

CASE II.—W. B., white, aged sixty-nine years, a printer. Had been an inmate of an insane asylum. There was no history of rheumatism; he denied syphilis; he had smallpox at thirty-nine years. He complained of dyspnœa on exertion, pain, and cough. He also was maniacal.

Physical Examination. The left subclavian artery and its branches pulsated more weakly than the corresponding arteries to the right. The carotids were equal. There was distinct downward tracheal tugging and suprasternal pulsation. The left ventricle was dilated, and there was a systolic murmur transmitted into the vessels of the neck. The fluoroscope showed a prominence to the left of the aorta, which had expansile pulsation. At the autopsy there was atheroma of the aorta with tortuosity of the descending portion. There was no aneurism and no dilatation.

CASE III.—J. H., white, aged fifty-seven years, machinist. He denied rheumatism and syphilis, but had had heart trouble for ten years. He complained of pain in the precordial region and dyspnœa.

Physical Examination. There was a receding pulse, with Traube's and Durosiez's signs. There was suprasternal pulsation and doubtful tracheal tugging. The left ventricle was considerably enlarged. There

was the double murmur of aortic regurgitation. There was no accentuation of the aortic second sound. The X-ray showed the characteristic pulsating prominence to the left of the aorta. A diagnosis was made of insufficiency of the aortic valves, dilatation of the aorta, and tortuosity.

CASE IV.—J. L., white, aged ninety-seven years, shoemaker. He denied syphilis and rheumatism. He suffered from dyspnœa on exertion; he had no cough and no pain.

Physical Examination. The arteries of the distribution of the left subclavian pulsated more freely than those of the right. The carotids were equal. There was suprasternal pulsation and slight tracheal tugging. The percussion note over the base of the sternum was impaired. There was general atheroma. The fluoroscope showed a pulsating mass to the right of the descending arch of the aorta. At the autopsy the aorta was found to be tortuous, and there was no aneurism and no dilatation.

CASE V.—N. McC., white, aged seventy-three years, stone-mason. Had had rheumatism and used alcohol to excess. He complained of dyspnœa on exertion and had a cough that was slightly metallic.

Physical Examination. The carotid and radial pulses were equal. There was distinct suprasternal pulsation and tracheal tugging. There was impaired resonance over the manubrium, and the second aortic sound was accentuated. The fluoroscope showed an abnormally broad aortic arch and pulsating prominence to the left of the descending arch.

CASE VI.—W. S., white, aged twenty-four years, a barber. He gave a history of rheumatism, and admitted alcohol in moderation, but denied syphilis. He had no dyspnœa and no pain. He had a hoarse but not metallic cough. The radial and carotid pulses were equal; there was distinct tracheal tugging; there was impaired resonance over the manubrium, and a faint systolic murmur heard over the base and transmitted into the vessels of the neck. There was no thrill. The fluoroscope showed a pulsating prominence to the left of the descending portion of the arch of the aorta.

CASE VII.—A. T., a laborer, had had rheumatism. He admitted alcohol in moderation, but denied syphilis. There was dyspnœa upon exertion and precordial pain, but no cough.

Physical Examination. The left radial pulse was smaller than the right. There was no tracheal tugging and no suprasternal pulsation. The fluoroscope showed a pulsating projection to the left of the descending portion of the arch of the aorta.

CASE VIII.—J. W., aged forty-three years, a negro laborer. Many of his family had heart disease. He had acute articular rheumatism at twenty-five years. On admission he had dyspnœa increased by exertion, cough and expectoration, and pain in the chest and epigastrium.

Physical Examination. The left radial was smaller than the right and seemed slightly retarded. The arteries were atheromatous. The heart was enlarged; there was a systolic musical murmur transmitted into the vessels of the neck, which subsequently showed a double murmur. There was slight tracheal tugging; a thrill was readily produced by pressure upon the subclavians and occasionally was spontaneous. The fluoroscope confirmed the great enlargement of the heart, the increased breadth of the arch of the aorta, and also showed a projection to the left of the descending portion of the arch that had expansile

pulsation. At the autopsy obliterative pericarditis was found, with hypertrophy of the heart, atheroma and marked dilatation, and tortuosity of the aorta at the junction of the arch and the vertebral column.

CASE IX.—H. W., white, aged sixty-eight years, a carpenter. He suffered from dyspnœa on exertion and gave a doubtful history of paralytic stroke.

Physical Examination. The left radial pulse was smaller than the right. The carotids were equal; the left pupil was slightly dilated. The heart was enlarged; there was a double murmur over the base; suprasternal pulsation and distinct tracheal tugging. Percussion resonance over the manubrium was impaired. The fluoroscope showed a pulsating projection to the left of the descending portion of the arch.

CASE X.—W. S., white, aged seventy years, a laborer. Had had rheumatism, but denied syphilis. He complained of severe cough, which was distinctly metallic in character.

Physical Examination. The left radial pulse was smaller than the right. There was distinct tracheal tugging; impaired resonance over the manubrium. The fluoroscope showed a distinct bulging to the left of the descending portion of the arch of the aorta which had expansile pulsation.

CASE XI.—S. McH., white, aged fifty-one years, a carpenter. Admitted syphilitic infection. At forty-three years had right hemiplegia.

Physical Examination. The pupils were equal; the carotids and radial pulses were equal. There was tracheal tugging and a faint suprasternal pulsation. The heart was not enlarged. The second aortic sound was not accentuated, but the first sound was loud. There was impaired resonance over the manubrium, and the patient had a hoarse, aneurismal cough, which had lasted seven years. The fluoroscope showed increased width of the arch of the aorta, and a projection to the left of the descending portion of the arch which had expansile pulsation.

CASE XII.—M. O., white, female, a domestic. She had been conscious of heart disease for two years

Physical Examination. Pupils were equal; the right radial pulse was slightly weaker than the left. There was expansile pulsation in the lower portion of the neck, which was apparently due to an atheromatous anomalous vessel. There was no tracheal tugging and no thrill. The heart was enlarged to the right and left. There was a systolic murmur transmitted into the vessels of the neck. The aortic second sound was greatly accentuated, and the pulmonic second was reduplicated. The percussion note over the manubrium was impaired. The patient had dyspnœa, but no pain and no cough. The fluoroscope showed dilatation of the ascending portion of the arch of the aorta that had expansile pulsation.

CASE XIII.—T. M., white, aged thirty-six years, laborer. He had had malaria, rheumatism, and syphilis, and admitted excessive indulgence in alcohol. For about two years he had had boring pain in the left chest, located usually in the sixth interspace, either just beneath the angle of the scapula or in the nipple line. It was worse after exertion and the sixth rib was tender.

Physical Examination. Pulsation in the left subclavian, brachial, and radial arteries could not be felt. In the right subclavian, brachial,

and radial arteries it was strong. The carotids were equal. There was a systolic murmur, heard best at the apex. The aortic second sound was greatly accentuated. There was slight paralysis of the left vocal cord (Dr. Gleason). The fluoroscope showed a distinct prominence of the descending portion of the arch, which had expansile pulsation. There was no dilatation of the aorta in any part. The patient is still alive, and the signs have not changed in eighteen months. It seems not impossible that in this case we are dealing with an actual aneurism, although the fluoroscopic picture is the same as that in the other cases.

CASE XIV.—R. W., white, aged forty-four years, a knife-grinder. He had had psoriasis for twenty years, and had used alcohol and tobacco to excess. He denied venereal disease. One year ago he had erysipelas.

Physical Examination. The pupils were equal; the left brachial and radial arteries pulsated more weakly than the right. The carotid pulses were equal. The heart was not enlarged. The aortic second sound was accentuated; there was no suprasternal pulsation, but distinct tracheal tugging. The fluoroscope showed increase in the width of the arch of the aorta, and a projection to the left of the descending portion which had expansile pulsation.

CASE XV.—M. B., white, female, domestic, aged seventy years. Had had rheumatism for many years. Had dyspnœa upon exertion, and had complained for twenty years of pain in the left side of the chest. There was a cough, which was not metallic in character.

Physical Examination. The radial pulses were equal; there was an anomalous vessel crossing the anterior portion of the neck, which was atheromatous. There was no tracheal tugging. The heart was slightly enlarged, and there was a double murmur over the base, the systolic element being transmitted into the vessels of the neck. The fluoroscope showed dilatation of the ascending portion and a projection to the left of the descending portion of the arch of the aorta which had expansile pulsation.

CASE XVI.—J. T., white, aged sixty-one years, broom-maker. Had malaria at sixty-six years; pleurisy at forty-five years; several attacks of asthma. He denied syphilis. He complained of cough and moderate expectoration.

Physical Examination. The right carotid and subclavian arteries and their branches pulsated more vigorously than the corresponding vessels on the left. There was no tracheal tugging, but a powerful suprasternal pulsation. The heart was slightly enlarged. There was a systolic murmur transmitted into the vessels of the neck, and the aortic second sound was accentuated. The fluoroscope showed dilatation of the ascending portion of the arch of the aorta and distinct prominence of the descending portion of the arch, with expansile pulsation.

CASE XVII.—P. D., white, stone-cutter. Had used alcohol to excess; he never had rheumatism, and denied syphilis. There was moderate dyspnœa, pain in the region of the heart, and a cough which was not metallic in character.

Physical Examination. The pupils were equal and of the Argyll-Robertson type. The right pupil was smaller than the left. The carotid and radial pulses were equal. There was a thrill in the vessels of the neck upon pressure. There was suprasternal pulsation and distinct tracheal tugging. The heart was slightly enlarged to the left;

there was a soft systolic murmur at the apex and base, and a harsh systolic murmur at the base. There was also a harsh systolic murmur in the vessels of the neck. The aortic second sound was not accentuated. The fluoroscope showed dilatation of the ascending portion of the arch of the aorta, and a projection to the left of the descending portion which had expansile pulsation.

CASE XVIII.—C. H., white, female, domestic, aged eighteen years. Patient had recently had acute articular rheumatism.

Physical Examination. The carotids and radials were equal. There was suprasternal pulsation, but no tracheal tugging. The heart was not enlarged; the second aortic sound was greatly accentuated. The fluoroscope showed that the ascending portion of the arch was increased three-fourths of an inch in width, and to the left of the descending portion there was a shadow that had expansile pulsation.

In summarizing these cases we find that the inequality of the pulses was present in ten and not present in eight cases; that in nine of the ten cases pulsation was more vigorous on the right than on the left side. Inequality of the pupils was present in three cases; in one of these there was reason to believe that it was due to some nervous condition, as the pupils showed the Argyll-Robertson phenomenon. Suprasternal pulsation was present in twelve cases and absent in six. Tracheal tugging of distinct downward character was present in eleven and absent in seven cases. A systolic thrill was felt in the vessels of the neck, either with or without slight pressure, in nine cases, and it could not be elicited in nine cases. A systolic murmur usually transmitted into the vessels of the neck was present in eleven cases, but was not heard in seven cases. Twelve of the cases had dyspnœa; in one of these there were physical signs of emphysema and chronic bronchitis; in the others it was probably due to the cardiac condition. Many of these patients were old, and nearly all of them had atheromatous arteries. Precordial pain was present in seven cases and was not complained of in eleven cases. In many of the cases complications in the form of valvular heart disease were present. In one case there was actually an aneurism of the innominate artery. The degree of atheroma and the age of the patient also modified the symptoms, and the extent of the tortuosity must vary considerably in different cases.

It is difficult from this study to give a clear picture of the symptomatology of the condition of atheroma of the aorta leading to tortuosity of the descending portion of the arch. It may at least be said that in those conditions in which atheroma and tortuosity exist, certain of the physical signs, particularly tracheal tug and inequality of the radial pulses, which have been supposed to be strongly suggestive of aneurism, are not uncommon, and that they are associated with other signs and symptoms, such as murmur, thrill, suprasternal pulsation, inequality of the pupils, accentuation of the aortic second sound, that are also found in aneurism of the arch. Probably it would be justifiable to suspect

tortuosity in cases in which there is inequality of the radial pulses, slight tracheal tugging, dyspnœa, and in which the symptoms are stationary for a considerable period of time and the fluoroscope shows a projection to the left of the descending portion of the arch of the aorta that has expansile pulsation.

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THE OCCURRENCE OF THE BENCE-JONES ALBUMIN IN A PLEURITIC EFFUSION.

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THE peculiar albumose-like body which has been named after Bence-Jones, who first gave it serious research, has during recent years demanded much attention. It is found in the urine of patients suffering from malignant bone disease, in particular either multiple myeloma or sarcomatosis, and also in the blood and bone-marrow of these conditions. Formerly it was thought to occur in osteomalacia, but it is now recognized by more modern methods of examination that what had been previously designated as osteomalacia is in reality multiple myeloma. The uniting of these facts has made the urinary examination of the highest importance in the diagnosis of obscure bone disease, especially those in which there is an absence of metastasis and several bones become simultaneously the seat of the new-growth. An albumose resembling the Bence-Jones body has been described by R. Fleischer[1] as occurring in normal bone-marrow, but further research along this line by the more modern methods of the separation of various albumoses seems to be necessary. The substance has also been encountered in the urine of animals the subjects of experimental pyrodin anæmia. To these findings I am now able to add to the reported series a case in which the Bence-Jones albumin could be demonstrated in large quantities in the pleuritic effusion of a patient suffering from multiple neuritis associated with extreme tenderness of the ribs, while it was absent from the urine. So far as I am able to discover this is a new observation, and one which I hope will cast some light on the much-discussed question of the origin of this substance. Although the

pleuritic effusion was very slightly purulent, yet in the literature there can be found only the vaguest references to the occurrence of "albumose" and "peptone" in pus and pathological pleuritic fluids. My case is the first to occur in the wards of an insane hospital, and the patient was the subject of an extensive multiple neuritis associated with a peculiar mental condition, of which the prominent symptoms were a profound memory defect associated with extensive fabrications. The clinical history is given in detail in order that the reader may judge of the varied symptom-complex under which this body may be demonstrated.

Polyneuritic delirium (Korsakow's disease); alcoholic excesses; multiple neuritis; extreme tenderness of the ribs; pleurisy with effusion; albumose in the pleuritic fluid; urine negative; no anæmia. The patient was an Englishman, aged forty-two years, of whose personal history but little is known, except that for several years he drank heavily and was frequently intoxicated. He was admitted to the Worcester Insane Hospital on October 29, 1902, with a certificate stating that the onset of his trouble had been several weeks previously, that he was in poor bodily health, and in a state of complete mental confusion. On admission to the hospital he was immediately placed in bed on account of his weak condition, as he was scarcely able to stand. There was a profound delirium with disorientation, prominent memory defect for both recent and remote impressions, and extensive fabrications which he constantly utilized to fill up the gaps in his memory. The mental condition has remained unchanged since admission. He was occasionally noisy at night.

The physical examination showed the following:

A middle-aged, fairly well-developed but poorly nourished man; weight, 130 pounds. Tongue covered with a brownish coating. The pupils are equal and react promptly to light and accommodation. There is a tendency to nystagmus in the horizontal movements of the eyes. Knee-jerks slightly exaggerated on the right, the left about normal. The tongue protrudes in the median line and has a fine tremor. There is a tremor of the lips when the teeth are shown. The grasps are very weak, and the muscular power of the extremities shows considerable weakness of the extensor muscles, the feet drooping as the patient lies in bed. He is unable to stand without support and complains of feeling dizzy. The examination of the chest shows a marked lack of expansion on the left side, with dulness in the region of the lower lobe, almost complete absence of breath sounds, and diminished vocal resonance. Pulse 100 and weak. Arteries everywhere thickened. Hands and feet cold. The urine showed a trace of albumin, diminished indican, and a few hyaline casts. Stools daily and regular. Occasionally the evening temperature rose to 100°. He gradually grew weaker, with continued fever and increasing delirium. On November 14th there was marked dyspnœa, and examination of the lungs showed complete dulness in the lower part of the chest, with diminished vocal fremitus. The chest was aspirated and about 1500 c.c. of a clear, amber fluid withdrawn, which resulted in considerable relief of the dyspnœa. On November 17th the knee-jerks were found to be absent

and the muscles showed considerable wasting, although the electrical reactions were normal. The fever continued, occasionally reaching 101° F., and on December 1st there was a return of the dyspnœa. The physical examination of the lungs showed the same condition as previously. The knee-jerks were still absent and the atrophy of the leg muscles more marked, the circumference of the left leg being from 1 cm. to 2 cm. less than on the right. There was some pain and tenderness over the nerve trunks. The left pleural cavity was again aspirated on February 10, 1903, and about 400 c.c. of a clear fluid withdrawn. This was repeated on March 2d, and 1800 c.c. of an amber serum aspirated. From the time of admission up to May, 1903, the patient lost twenty pounds in weight. The mental and physical condition remained unimproved. On May 26, 1903, 1050 c.c. of fluid was again aspirated from the left side of the chest, and it was this fluid which I had the opportunity of examining.

Analysis of the Pleuritic Fluid. Amount, 1050 c.c.; color, greenish-yellow and semi-opaque; specific gravity, 1027; odor, none; reaction, neutral. The amount of sediment was small. On standing in a tall, cylindrical litre graduate for twelve hours it did not occupy more than ½ cm. The microscopic examination showed a fair number of normal pus cells, the absence of cholesterin crystals, and a few blood cells (contamination from aspiration with the trocar).

Fibrin was absent. There was no spontaneous coagulation. On heating there was a fairly firm coagulation of serum albumin and globulin. On adding 10 per cent. acetic acid in the cold there resulted a fair precipitate of nucleoproteid. A portion of the fluid was made slightly acid with acetic acid and the other proteids removed by boiling. The filtrate was slightly cloudy, which cloudiness could not be removed by continued filtration. Tests for serum albumin and globulin and nucleoalbumin showed all to be entirely absent.

On the addition of a few drops of nitric acid to the filtered fluid there resulted a heavy white precipitate, not soluble in excess of the acid, but which completely cleared up on heating and reappeared on cooling with the simultaneous appearance of the xanthoproteic reaction. The same reaction was obtained with sulphosalicylic acid. The amount of this albumose-like body was large and reached 4 per cent. in the Esbach albuminometer. As this substance was undoubtedly an albumose, its further investigation was decided upon.

Repeated examinations of the urine showed it to be free from both albumin and albumose. Further examination of the patient revealed extreme tenderness on pressure over the sixth to the tenth ribs on the left side, with some crepitation under the skin, but without any herpetic eruption. No nodules or swellings were felt along the line of the ribs, and the long bones of the legs and arms were free from pain, both to pressure and percussion. A few days later this tenderness disappeared. There was no evidence of anæmia, and the blood examination showed: Hæmoglobin, 100 per cent.; erythrocytes, 4,800,000; leucocytes, 7640.

Reactions of the Native Fluid. A portion of the fluid was diluted with water, and 10 per cent. acetic acid added to a slightly acid reaction. There resulted a moderate precipitate of nucleoproteid. The fluid was then heated upon the water-bath until the native albumin was coagulated and the rather heavy coagulation filtered off by aid of an exhaust

filter. The slightly cloudy filtrate was found to be free from serum albumin and globulin and nucleoproteid. This native fluid responded to the following reactions:

On heating there resulted a slight milkiness at first, but the fluid rapidly became opaque. As the boiling point was reached white flakes appeared in a clear liquid, which rapidly settled to the bottom of the tube as a fine, flocculent precipitate, which did not increase on cooling. Heller's test was positive, a broad white ring.

On the addition of a few drops of nitric acid a heavy white precipitate formed in the cold, which did not dissolve in excess of the acid, but almost completely disappeared on boiling (the fluid at the same time turning yellow), and reappeared on cooling as a granular, yellowish precipitate. This turned to orange on the addition of a strong alkali (xanthoproteic reaction).

Hydrochloric acid produced the same result, with the exception that on heating the precipitate at first grew more dense and then disappeared as the boiling point was reached. Sulphuric acid produced a similar reaction. With phosphoric acid there was a white precipitate in the cold, which did not disappear on heating. Sulphosalicylic acid produced a heavy white precipitate in the cold, which disappeared on heating and reappeared on cooling. It was noted that the reaction was more marked in this case than with any other acid reagent.

On the addition of acetic acid even in excess the solution remained clear, both in the cold and on heating. Tannic acid caused a heavy, yellowish-brown precipitate, which grew more dense on heating. Both picric acid and Esbach's reagent caused a heavy yellow precipitate, which did not disappear on warming.

The addition of phosphotungstic acid in the presence of a few drops of hydrochloric acid caused a heavy, white precipitate, which curdled on heating, but did not disappear. Acetic acid and potassium ferrocyanide produced a heavy precipitate, which grew more dense on heating.

On the addition of Lugol's solution no precipitate was obtained. Alcohol precipitates the substance as a flocculent mass readily soluble in very dilute ammonia or sodium-carbonate solution.

The resulting solution gives the albumose reactions with sulphosalicylic and nitric acids. Millon's reaction, intense brick-red; Adamkiewicz reaction, deep purplish-red; xanthoproteic reaction, strong; Molisch's reaction, the sulphuric acid turns green almost immediately, and on standing a short while a purplish-red ring forms at the junction of the acid and the fluid; biuret reaction, intense purplish-red reaction with the native fluid.

To another portion was added phosphotungstic acid and a few drops of HCl, the resulting heavy, white precipitate washed with absolute alcohol, dissolved in weak sodium-hydrate solution, and heated until it became colorless. On the addition of a few drops of a weak copper-sulphate solution to this fluid a purplish-red biuret reaction resulted. The solution remained clear on the addition of an excess of a saturated sodium-chloride solution. On heating there was a very slight turbidity, but no precipitate resulted. This turbidity could not be removed by filtration. In the presence of a few drops of 10 per cent. acetic acid, however, a saturated salt solution caused a heavy, white precipitate, which did not disappear or grow less on boiling. Saturation with sodium

chloride in substance or with rock salt did not cause a precipitate at room temperature until the point of saturation was reached. This was hastened by warming. Saturation with magnesium sulphate in the presence of acetic acid caused a complete precipitation of the substance.

The addition of twice the amount of a saturated solution of ammonium sulphate to the neutral fluid caused a complete precipitation of the body. The substance was not dialyzable and could not be precipitated by dialysis, even from salt-containing solutions.

After coagulation by heat the coagulum went into clear solution on warming with distilled water to which was added a little sodium carbonate or acetic acid. From this solution it could be precipitated by alcohol. The substance could not be precipitated from its acid solutions on neutralizing with ammonia or even when it was added in excess. The neutral solution remained clear, even on largely diluting with water. On passing CO_2 through the neutral solution no precipitate resulted. Gravimetrically, the amount of the albumose was 4.36 per cent. The sulphur test was marked: a dark brown color which rapidly became black, and, finally, a black precipitate.

Coagulation Temperature. There was a slight beginning opalescence at 43.5° C., which became more marked at 50° C. The solution began to be turbid at 65° C. and opaque at 67° C., so that the bulb of the thermometer could scarcely be seen. Flocculi began to separate at 69° C., and at 69.5° C. these flocculi rapidly settled to the bottom of the tube and assumed a granular appearance.

Crystallization. On slow precipitation by gradual heating the substance became granular in appearance, and under the microscope could be found a large number of gleaming white oval and round spherolithe resembling leucin. In many of these could be seen concentric and radiating lines. These crystals were easily soluble in dilute hydrochloric and acetic acid and in sodium carbonate. No crystals could be obtained when the native solution was allowed to stand for several days or to slowly evaporate. A portion was precipitated with a saturated solution of ammonium sulphate and evaporated over calcium chloride, but crystallization failed to take place.

Reactions of the Isolated Substance. To another portion of the fluid there was added twice its volume of a saturated solution of ammonium sulphate. The resulting heavy precipitate was filtered off, dissolved in hot water, and dialyzed for some time. Even on prolonged dialysis, however, it was found impossible to get rid of all the salt. The most delicate tests failed to show that the substance was dialyzable, and neither was it precipitated as the amount of salt grew less. The solution had a slight opalescence and was neutral in reaction.

It responded to the same color and precipitation tests as the native substance and also crystallized on slow heating, the crystals again resembling leucin. The reaction to various salts both in solution and in substance was likewise identical, and the coagulation point was at the same temperature. Evidently the trace of ammonium sulphate that was present and could not be removed by dialysis did not influence the various reactions, crystallizations, or coagulation point.

The following reactions, however, may be especially noted:

No precipitate occurred either on largely diluting with water or on the passage of CO_2 through the liquid. On adding Lugol's solution,

no precipitate. On adding an equal quantity of a saturated sodium-chloride solution there is no precipitate in the cold and only a slight opalescence on heating, which cannot be removed by filtration. If the same process be repeated with the addition of a few drops of acetic acid there results a heavy, flocculent precipitate in the cold, which grows more dense on heating. Saturation with sodium chloride in substance or with rock salt causes a partial precipitation, which becomes complete on the addition of a few drops of acetic acid. If coagulated by heat the coagulum dissolves readily, forming a clear solution on the addition of a few drops of acetic acid or of a saturated solution of sodium carbonate. It can be precipitated from either of these solutions by neutralization with sodium carbonate (if acid) or with acetic acid (if alkaline), as the case may be.

Elementary Analysis. For elementary analysis and digestive experiments the albumose was precipitated with alcohol, the resulting precipitate washed several times with 95 per cent. alcohol, then absolute alcohol and ether, and dried over calcium chloride. The substance purified in this manner was almost pure white and responded to the following reactions when dissolved in dilute ammonia, in which it was readily soluble: Biuret reaction, rose-red; Millon's reaction, intense vermillion-red; xanthoproteic reaction, marked; Molisch's reaction, purple at junction, the acid turning green; Adamkiewicz reaction, marked purplish-red; sulphur reaction, moderate; nitric acid, white precipitate, which completely disappears on heating and reappears on cooling; sulphosalicylic acid, same as with nitric. With the pure dried substance Lieberman's reaction gave a deep lilac color. The amount of nitrogen was 15.4 per cent. Both phosphorus and the xanthine bases were absent, but there was a trace of iron. Sulphur was present.

On warming a portion of the substance for an hour with dilute hydrochloric acid there was obtained a reducing body which gave marked reactions with Fehling's and Nylander's solutions. With phenylhydrazin and sodium acetate there was obtained an osazone which crystallized in long and rather broad, yellow needles, but in too small an amount to determine their melting point. The hydrochloric acid phloroglucin test was negative.

Behavior on Digestion. On digestion with trypsin in a slightly alkaline medium at 40° C. for forty-eight hours the substance went into complete solution. No neutralization precipitate was obtained with acetic acid. The test for protein chromogen with bromine-water was well marked, and there was a large yield of tyrosin and but little leucin. The leucin and tyrosin were identified by their crystalline form and the latter in addition by its intense Millon reaction. I regret that my material was not sufficient for peptic digestion.

From the above reactions we can conclude that the pleuritic fluid contained the so-called albumose of Bence-Jones, which was formerly thought to occur only in malignant bone disease, but which later investigations have shown to be present in other conditions.

There have been reported cases of albumosuria associated with various neuritic disorders or with vague symptoms referable to the nervous system, and some of these at least seem to have been free from any bone or marrow disease.

Senator's case[3], in addition to a multiple sarcomatosis, had central hypoglossus and trigeminal paralysis, with great weakness and sensitiveness of the left peroneal and tibial nerves. The tongue was especially involved. The autopsy showed multiple myelogenous round-celled sarcoma of the ribs, double-sided fibrinous pleurisy, and chronic parenchymatous nephritis with amyloid degeneration. The brain showed no abnormality, and the medulla exhibited only an irregular distribution of the nuclei. In the urine, which was examined by Rosin,[3] large quantities of albumose could be demonstrated.

In Wright's case[4] there was numbness of the feet, double vision, and strabismus. Hamburger's first case[5] had frequent attacks of facial neuralgia. Boston's case[6] suffered from gastrointestinal catarrh and peculiar nervous phenomena, attacks of obstinate constipation, radiating pains, vomiting, œdema, frequent attacks of urticaria, various paræsthesias, loss in weight, and marked anæmia. It is merely mentioned that the Bence-Jones body was present in the urine and that its elimination was intermittent in character. No chemical details are given. Anders and Boston[7] later report three cases in which the Bence-Jones albumose was found in the urine. Saturation with sodium chloride in neutral solutions caused a precipitate in but a portion of the substance, thus differentiating it from protoalbumose and heteroalbumose. The first case had cranial and facial neuralgia, the second suffered from various paræsthesias and neuralgic pains in the lower limbs, while in the third case there was paralysis of the left side of the face. The authors contend that the body is more or less closely allied to peptone, globin, histon, and the digestive albumoses.

Since Simon collected the literature in 1902 there have been reported additional cases of Bence-Jones albumosuria associated with symptoms referable to the osseous system or to the blood. One of these (Jochmann and Schumm) is a further report of the urine and post-mortem findings of a case already published. In Simon's case[8] there was pain along the course of the eighth and ninth ribs, with extreme tenderness, and followed shortly afterward by a tumor mass, the size of a lima bean, near the sternal end of the eighth rib. There was marked anæmia and great loss of weight. For three months before death there existed extreme pain of the left thigh, and five days before exitus spontaneous fracture of the left femur occurred. Death ensued a year after the onset of the first symptoms, but the partial autopsy that was allowed revealed no abnormality of the ribs. The diagnosis of multiple myeloma was made at the beginning of the disease on the basis of the urinary findings. There was polyuria, low specific gravity, acid reaction, and the sediment was free from casts. Heated to 55° C. the urine yielded a heavy, white precipitate, which became a little clearer on boiling, to grow more turbid on cooling, and which again became clear

on the addition of a few drops of acetic acid. Nitric acid yielded a precipitate which completely dissolved on boiling and reappeared on cooling. The urine gave a strong biuret reaction. By Esbach's albuminometer the amount of proteid was 0.27 per cent. The author undertook an elaborate chemical study of this substance and concluded that it was closely related to the globulins, if, indeed, it is not a globulin itself. The body showed many points of resemblance with Nöel-Paton's crystalline globulin and with Marcus' water-soluble globulin. He argues strongly against the purely albumose nature of the substance.

In Gutternik's and de Graff's case[9] the albumin could be crystallized, and the authors refer to it as a crystalline urinary albumose. This, including Nöel-Paton's and my own, makes three cases in which the substance could be obtained in crystalline form.

Vickery's case[10] was one of albumosuria occurring in the course of a pernicious anæmia without any symptoms or abnormalities referable to the bones. The urine was examined by Wood. By the heat test there formed an abundant precipitate which dissolved on boiling and reappeared on cooling. No attempt was made to further identify the nature of this albumose-like body.

Jochmann and Schumm[11] report the case of a woman, aged thirty-seven years, which, in a preliminary communication, was called osteomalacia,[12] but later, after an elaborate study, the diagnosis was changed to multiple myeloma. During life the case was submitted to a careful X-ray examination. The Bence-Jones albumose was present in the urine, and Schumm gives an extensive chemical study of the body, arriving at the conclusion that it is not a true albumose, but stands midway between albumose and albumin. He was able to demonstrate the presence of an albumose-like body in the blood, which resembled deuteroalbumose and did not give the histon reactions. Barr[13] reports a urine that contained a proteid which coagulated at 60° C. and could be precipitated by mineral acids, but was soluble in excess. It could be precipitated by ammonium sulphate, but not by magnesium or sodium sulphate. The amount of proteid varied from 1.2 per cent. to 2.4 per cent. The reactions given, however, do not prove the identity of this substance with the Bence-Jones albumin.

In Milroy's case[14] there was a knotty swelling under one of the ribs which appeared like a new-growth. The urine, when made weakly acid with acetic acid, gave a precipitate by warming at 52° C., which thickened to a coagulum and dissolved largely at 95° C. It reappeared again on cooling. The amount of this proteid was 0.54 per cent. The urine gave the biuret, Millon's xanthoproteid, and sulphur reactions, but phosphorus was absent. Saturation with sodium chloride or with magnesium sulphate gave a very slight precipitate. Nitric acid

gave a precipitate in the cold which dissolved on warming and reappeared on cooling. By repeated precipitation with ammonium sulphate, the behavior of the substance on warming was not changed. If the urine was warmed rapidly to 100° C. the substance did not appear on cooling. When the urine was saturated with sodium chloride and then heated the entire mass coagulated. It was impossible to crystallize the proteid. The substance as a whole appeared to resemble a deuteroalbumose, but behaved differently on warming.

In Simon's case of læukemia[15] with atypical blood findings (absence of eosinophilic leucocytes) the Bence-Jones albumin could not be demonstrated in the urine, even on repeated examinations.

In addition to the above, in the urine of a case of puerperal delirium associated with an acute parenchymatous nephritis with occasional hemorrhagic exacerbations, and having a freezing point varying from 0.15° C. to 1.6° C., a high percentage of albumin, many casts, and a low chloride and urea elimination, I found large amounts of an albumose which gave the following reactions: Nitric acid, heavy, white precipitate in the cold, disappearing on heating and reappearing on cooling; sulphosalicylic acid, the same as with nitric acid, but more marked; biuret reaction, rose-red; sulphur test, marked; Molisch's reaction, marked; xanthoproteic reaction, strong; Millon's reaction, marked.

The addition of acetic acid causes no precipitate, either in the cold or on heating. With alcohol there arises a white precipitate which is easily dissolved in weak sodium carbonate, and the resulting solution gives the biuret reaction. My investigations on this case are not sufficiently mature to warrant further detail.

The proteid substance found in the urine of the various cases cited was of an albumose nature, yet the finer chemical details differ somewhat. They were all associated with some profound metabolic disturbance, manifested clinically by either abnormal blood states, new-growths in the bones, or by various trophic or neuritic disorders. The reaction to mineral acids (especially nitric) seems to have been identical in all the cases. The slight variations occur in their physical states or in their composition on elementary analysis, but even the latter shows a remarkable resemblance, as will be seen from the following analyses of various observers:

ELEMENTARY COMPOSITION.

Observer.	N.	S.	P.	Fe.
Bence-Jones	15.08	Present.	Present.	.....
Kühne	16.55	"	.....	.....
Nöel-Paton	16.06	"	Absent.	.....
Neumeister	15.55	"	"	Trace.
Simon	15.12–15.26	"	"	.....
Coriat	15.4	"	"	Trace.

The presence of the carbohydrate group is a prominent characteristic and is probably related to the hexoses, as it failed to give the reaction for pentoses with the phloroglucin-hydrochloric acid test. Pick,[16] moreover, speaks of his fraction III. as containing a carbohydrate molecule and calls it a glycoalbumose, yet this group was present in his other secondary albumoses, although in a smaller amount. This fraction resembles my substance by its positive Molisch test, yet, on the other hand, differs from it in various precipitation and color reactions.

The Bence-Jones body is also to be differentiated from various other proteids and albumoses, and the essential points may be stated as follows:

Histon is an albumose-like body found by Kossel in the red blood corpuscles of geese and also in febrile and leukæmic urine. It does not exist as such in the cells, but in combination with another body, presumably nuclein. It contains 16.7 per cent. to 17.93 per cent. nitrogen, according to various observers. When treated with alcohol it becomes insoluble in sodium carbonate or dilute acids, it is precipitated by CO_2 from neutral solutions and by ammonia from acid solutions, and is insoluble in excess of the reagent.

Heteroalbumose is precipitated by saturation with sodium chloride in neutral solutions, yields but little tyrosin on tryptic digestion, and contains 17.98 per cent. nitrogen. It is also precipitated on dialysis and does not contain a carbohydrate group in its molecule.

Protoalbumose is dialyzable to some extent, but is not precipitated on dialysis. It is non-crystalline and gives only a faint Adamkiewicz reaction, does not contain a reducing body, and is only partially precipitated by sodium chloride from its neutral solutions.

Serum globulin coagulates at 75° C., is precipitated by diluting its salt-containing solutions with water, on dialysis after precipitation with various salts, by passing a stream of CO_2 through its solutions, by half saturation with ammonium sulphate, or by complete saturation with sodium chloride. According to Mörner,[17] it yields a reducing body on boiling with dilute acids.

With the ordinary albumins it has nothing in common. Peptone is not precipitated by nitric acid or on saturation with ammonium sulphate.

Deuteroalbumose is an anticompound, and does not yield any amido-acids on tryptic digestion. It is precipitated by sodium chloride only on the addition of acetic acid, and does not give the nitric-acid reaction except in the presence of an excess of salt, and is dialyzable to some extent.

The substance in my case gave all the ordinary albumose reactions, but unlike the albumoses it could be crystallized, and yet resembled the colloids in not being dialyzable, while protoalbumose and hetero-

albumose are dialyzable to some extent. It seems to have existed in the fluid in a peculiar physical condition, as in Nöel-Paton's crystalline globulin, or in Cramer's three cases of emulsion albuminuria.[18] Even on coagulation by both heat and alcohol it was capable of being easily dissolved by very dilute acids or alkalies and forming a clear solution, while histon is insoluble under the same conditions. Furthermore, unlike histon, it could not be precipitated by ammonia from its acid solutions. The Molisch reaction was positive, and the carbohydrate group present seemed to be related to the hexoses, while both protoalbumose and heteroalbumose have no carbohydrate group in their molecule. It also differs from protoalbumose and heteroalbumose in being precipitable by saturated solutions of sodium chloride only in the presence of acetic acid. The coagulation temperature was a definite one. Both phosphorus and the xanthin bases were absent, proving that the substance had no relation to the nuclein group. That it is a hemibody is shown by the large yield of tyrosin on tryptic digestion, while deuteroalbumose, the substance which the Bence-Jones albumin most resembles, is an antibody and does not yield the amido-acids when digested with trypsin. The behavior on boiling has been noted differently by various observers; with some, the substance almost completely dissolved; in Nöel-Paton's case there was complete coagulation, while in Simon's and my own case there was no evidence of clearing, even when the boiling point was long sustained, but it is noteworthy that in both cases the coagulum was capable of easy solution.

The exact nature and origin of the Bence-Jones substance has been the subject of much contention. Simon believes that the body is closely related to the globulins; Huppert looks upon it as a heteroalbumose; while Kühne and Chittenden found that on elementary analysis the body resembled heteroglobulose, and they suggest that it may arise from serum globulin. Magnus-Levy, on the other hand, did not believe that it belonged to the globulins, on account of its behavior to magnesium sulphate.

There is no reason to believe that the substance is formed in the kidneys, but it is eliminated by these organs when abnormally present in the blood; yet here again direct proof is wanting as to the origin of the substance in that medium. Schumm was able to demonstrate in the blood of his case a body which resembled a deuteroalbumose. Following the introduction of the isolated body into the blood of animals it rapidly appears in the urine. The presence of an albumose-like substance in normal bone-marrow proves nothing, but it is interesting to note that both Wood and Ellinger have found, in the affected portion of the marrow in malignant bone disease, a proteid which gave all the albumose reactions. That the substance arises as the result of peptic or tryptic action upon ingested albumins, and then passes into the

blood stream, where, as a foreign substance, it is eliminated by the kidneys, is not to be thought of, because, as has been previously shown, the Bence-Jones albumin possesses nothing in common with the ordinary digestive albumoses. Simon claims that it is probably derived from the common albumins of the blood, in all probability the serum globulin, through the enzymotic action of the plasma cells of the bone-marrow, in cases of myeloma, and from thence passes into the urine. This, in the light of my own case, is at least very suggestive if not convincing. Up to this time, however, no case had been reported where the Bence-Jones albumin could be demonstrated in a pleuritic effusion, its occurrence being confined to the urine, blood, and bone-marrow. As a small amount of pus cells were contained in the pleuritic fluid of my case, it must necessarily have been of inflammatory and probably also of bacterial origin, as shown by the clinical course of the disease. If we look upon the pleuritic effusion as being in reality an exudate from the blood, the origin of this substance is probably from the same source, and can be referred to the action of enzymes contained either in the leucocytes or in the micro-organisms upon either the serum albumin or serum globulin. The process is really a digestive one upon the ordinary albumins of the exudate, which have the same composition and ratio as the proteids of the blood, but which, as the result of this digestive action, have in part been changed to an albumose having all the characteristics of the Bence-Jones body, and existing in the fluid in an unusual physical state. But whether this action is principally upon the serum albumin or serum globulin, or both, cannot be definitely decided at present. In confirmation of the above, Fermi and Pampersi[19] have shown that bacteria contain a proteolytic enzyme whose action upon proteids resembles that of trypsin. According to Austin,[20] the prolonged action of micro-organisms on nucleins, fibrin, and probably on other proteids, produces protoalbumoses and heteroalbumoses and secondary albumoses, A, B, and C, according to Pick's scheme, but no peptone. These albumoses simulate those produced by trypsin.

In the light of this recent work it is easily conceivable how an albumose having all the peculiar characteristics of the Bence-Jones body could arise in this manner from the native blood proteids as they were exuded into a pathological pleuritic fluid. The medullary theory of the origin of the substance cannot be sustained, because there is not sufficient bone-marrow, even if all were involved, to account for the large amounts of albumose present in the various cases. In my case, at least, the rib tenderness was not sufficiently prominent to be suggestive of malignant bone disease.

In conclusion, it may be stated that the substance in my case was pre-eminently of an albumose nature, although probably not a true albumose, and having all the characteristics of the Bence-Jones albumin,

but existing in the pleuritic fluid in an unusual physical state. It evidently was not absorbed and did not pass into the blood stream, as not even a trace of the substance could be detected in the urine. Furthermore, it seems to be a derivative of the digestive action of either the leucocytes or the bacteria upon the proteids in the effusion which are derived directly from the blood. Its origin can probably be traced to serum globulin rather than to serum albumin, as it possesses many features in common, both in reaction to various reagents and its composition on elementary analysis to Marcus' water-soluble globulin. The nitrogen content is about the same in both; neither are precipitated by dialysis or on the passage of CO_2 through their solutions, and both give the sulphur, biuret, xanthoproteic, Adamkiewicz, and Molisch reactions.

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MULTIPLE MYELOMA (MYELOMATOSIS), WITH BENCE-JONES PROTEID IN THE URINE.

(MYELOPATHIC ALBUMOSURIA OF BRADSHAW, KAHLER'S DISEASE.)[1]

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WITH A REPORT ON THE CHEMICAL PATHOLOGY.

By R. Hutchison, M.D.,
AND
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The patient, J. T., aged fifty years, a stoker, came under my observation at the German Hospital in May, 1900, but I did not make the diagnosis of multiple myeloma until the following July, when I happened to examine the urine by the ordinary methods. From May to July the Bence-Jones proteid[2] in the patient's urine had been frequently, at one time daily, measured by Esbach's albuminimeter, but had been entered in the notes as ordinary albumin. The daily amount of urine was found to be about 2000 c.c., and it contained about 7 per mille of the proteid. The reactions were quite typical, just as those described by Bradshaw, Kühne, Bence-Jones, and others. The most characteristic are, I think, the following (that is, when the urine is acid):[3] Coagulation of the proteid at a much lower temperature (about 58° C.) than ordinary albumin, more or less solution of the precipitate at a higher temperature (*e. g.*, when the urine is boiled), and complete or almost complete solution on adding acetic acid to the boiling urine; after the precipitate has been partially redissolved by boiling, a characterisic reprecipitation should take place on allowing the urine to cool. What Dr. Bradshaw considered to be the spontaneous precipitation of the proteid in his case (noted also in some other cases) was likewise observed in the urine of the present case. The urine sometimes was turbid with this precipitate when quite freshly passed, and on these occasions, the reaction being always very acid, the turbidity could not have been due to phosphates.

Summary of the Present Case.

The patient, a rather fat man, aged fifty years, complained of rheumatoid symptoms, commencing, so he thought, about the end of the

[1] Founded on a communication to the Royal Medical and Chirurgical Society of London, March, 1903.

[2] On account of the contention by A. Magnus-Levy (Hoppe-Seyler's Zeit. f. Phys. Chemie., Strasburg, 1900, vol. xxx. p. 200) that the so-called Bence-Jones albumose is really an albumin, I have referred to it in this paper as Bence-Jones proteid. For the sake of brevity, however, I have sometimes spoken of "Bence-Jones albumosuria" when I meant to signify the presence of Bence-Jones proteid in the urine. It seems that precipitin experiments fail to solve this question of the nature of the Bence-Jones proteid. In fact, the so-called "biological method" fails to distinguish the Bence-Jones proteid from various other proteids of human origin. *Vide* Rostoski, Zur Kenntniss der Präcipitine, Verhandl. der phys.-med. Gesellschaft, Würzburg, 1902, vol. xxxv. pp. 30-32.

[3] The urine is nearly always acid in Bence-Jones albumosuria.

year 1899. About February, 1900, he began to suffer from pains in his loins and stiffness in the small joints of his hands. Soon afterward the upper part of his back began to bend, so that he always had a stooping attitude. Previously to this illness the patient had been strong, but as a young man had had gonorrhœa and a chancre on the penis. One of his sisters suffered from diabetes mellitus.

The urine of the patient was found to contain the Bence-Jones proteid. The daily amount of the urine was about 2000 c.c., and it contained about 7 per mille of the proteid as measured by Esbach's albuminimeter. By a more exact method (precipitation with alcohol, drying, and weighing) Dr. R. Hutchison found that about 15 grammes of the proteid were excreted daily. The reactions of the proteid were the typical ones described by Bence-Jones, Kühne, Bradshaw, etc.

For some time the patient's condition remained fairly stationary, and at first, by the use of local hot baths, massage, etc., the power of bending his fingers was improved. Afterward, however, the general weakness, cachexia, and anæmia greatly progressed, and gummatous disease of the tongue and over one rib made its appearance. Examination of the blood showed great anæmia and slight leucocytosis. On January 25th the patient died after copious hemorrhage from the intestines, which post-mortem examination showed to be due to chronic ulceration of the duodenum. The Bence-Jones albumosuria persisted to the last.

At the necropsy the bone-marrow of all the bones examined was found to be more or less affected by a diffuse sarcoma-like growth of rounded or polyhedral mononuclear cells—a form of "multiple myeloma" or "myelomatosis." There were no localized outgrowths projecting from the bones, such as have been noted in some cases of multiple myeloma, and no other parts of the body were invaded. In fact, as the neoplasm was strictly limited to the osseous system, no parts of it could be regarded as metastatic. The presence in the tumor cells of certain granules and globules of various sizes constituted a striking histological feature in the present case. Professor R. Muir, of Glasgow, who made a careful histological examination of the growth, concludes "that the tumor is formed by a special and characteristic type of cell, which is probably derived either from the neutrophile myelocyte or its predecessor; that this cell produces in its protoplasm in a granular form a substance which is closely allied to though not quite identical with the substance of the neutrophile granules; and that this substance is formed in excess, and may form larger granules by confluence of the smaller, the larger globules sometimes becoming free."

According to J. H. Wright's report on a case examined by him,[1] the principal tumor cells in the case in question appeared to be a variety of Unna's "plasma cells." Dr. J. M. H. MacLeod has kindly examined sections from the present case to compare the cells with Unna's plasma cells. He has used the special methods of staining for this purpose, and tells me: "Morphologically the cells resemble plasma cells in that they are polyhedral and their nucleus is placed excentrically. The nuclei, however, do not show the characteristic arrangement so gener-

[1] Johns Hopkins Hospital Reports, 1900, vol. ix. pp. 359-366. (See Case No. 13 in the Summary of Cases at the end of the present paper.)

ally found in plasma cells of five or six deeply stained chromatin bodies around the periphery."

The Chemical Examination of the Organs (Report Contributed by R. Hutchison, M.D., and J. J. R. Macleod, M.D.).

Up to the present time only three cases have been recorded in which a chemical investigation of the organs was made.

The first of these is by Ellinger,[1] but the clinical history of the case shows it to have been of an unusually acute nature, and the symptoms scarcely typical of myelogenous disease.

The tissues examined were a piece of infiltrated rib, blood, and some ascitic fluid.

In the case of the piece of rib, the following somewhat rough method was employed. The piece of rib was macerated with water, the resulting extract was filtered off, and to part of it was added some sodium chloride and acetic acid, whereby a precipitate was produced. In another part of the watery extract the point of heat coagulation was determined and found to be 40° C.

The author mentions that a piece of normal rib-marrow treated in the same way does not give these reactions.

In the case of the blood and ascitic fluid, a more reliable method was employed.

To each was added an excess of alcohol, and the resulting precipitate was allowed to stand several days. It was then filtered off and macerated with water. The resulting watery extract was then weakly acidified with acetic acid and boiled. The boiling fluid was quickly filtered, so as to separate all coagulated proteid, and the filtrate, at first quite clear, gradually became cloudy on cooling, and gave the reactions for Bence-Jones proteid.

The second case is recorded by Askanazy,[2] and is peculiar in that an examination of the blood during life, and of the lymphatic glands after death, showed it to be one of lymphatic leukæmia.

The tissues examined were marrow from the vertebræ and head of femur, lymphatic tumors, blood, pericardial and pleuritic fluids.

Two methods were employed in testing for the proteid. One of these consisted in making a watery or weak caustic soda extract of the tissue. This was then treated with acetic acid, so as to precipitate nucleoproteid, which was filtered off. In the filtrate the heat-coagulation point was determined. It was then boiled and filtered hot, the resulting filtrate being gradually cooled. The other method consisted in adding alcohol to the extract, collecting the precipitate, and macerating it with weak caustic alkali. The resulting extract was then treated with acetic acid to precipitate nucleoproteid, and to the filtrate from this were applied the tests for Bence-Jones proteid.

By means of these methods it was found that the Bence-Jones proteid was present in the marrow, but absent from the lymphatic tumors, blood, and pericardial and pleuritic fluids.

The alcohol method was employed in a sample of urine containing the albumose, and was found to give reliable results.

[1] Deut. Arch. f. klin. Medicin, 1899, Bd. lxii. S. 255. (See Case No. 9 in the Summary.)

[2] Ibid., 1900, Bd. lxviii. S. 34. (See Case No. 16 in the Summary.)

The third case is that of Kalischer,[1] and the tissues examined were marrow from the ribs and humerus. The examination was made by Prof. Löwy, but he could not find a trace of Bence-Jones proteid.

Methods Employed in the Present Case. In the present case a very complete chemical investigation was possible, as we were fortunate in securing not only portions of all the organs, but also large quantities of bone from various regions. Two methods very similar to those described by Magnus-Levy were employed to detect the body.

The bones were crushed into fragments in a quartz crusher, the other tissues being chopped up into small pieces. Each was then ground up in a mortar with distilled water and the resulting pulp allowed to stand twenty-four hours. The extract was filtered off and the remainder again pounded in the mortar with distilled water, the second extract, after filtration, being added to the first.

The resulting watery extract from each tissue was then carefully neutralized and divided into two equal parts. To *one* of these were added two volumes of a saturated solution of ammonium sulphate, and the mixture was allowed to stand twenty-four hours. The resulting precipitate was separated by filtration, washed with ammonium-sulphate solution, and suspended in water. After standing several days this suspension was filtered and the filtrate placed in a parchment dialyzer for several days in running water, and finally, for at least two days, in distilled water, which was frequently changed. After dialysis a precipitate invariably separated out (globulin), which was removed by filtration. The resulting filtrate was tested for Bence-Jones proteid. To the *second* portion of the neutralized watery extract twice its volume of spirit was added, and the mixture was then boiled (by which means the native proteids become coagulated, whereas the Bence-Jones proteid does not).[2] The mixture was then filtered, and the precipitate, after being washed with boiling 66 per cent. spirit, was suspended in ¼ to ½ per cent. ammonia and either left in this for several hours or heated on the water-bath, by either of which processes a solution of the albumose results, while the coagulated proteids remain unaffected.

The former of these methods we will designate the ammonium-sulphate method, the latter the alcohol method.

These two methods were applied to urine containing the substance in question. The resulting solutions gave the chief reactions for Bence-Jones proteid.

To serve as controls we also examined, by both methods, red marrow from the vertebræ of a healthy person, and, by the ammonium-sulphate method, red marrow from the ribs of a horse. *In neither of these was any trace of Bence-Jones proteid*[3] *found.*

Results. The following are the results obtained from the various organs and tissues:

Marrow of Bones. The marrow contained in these was apparently of two types—namely, a pink, pasty mass in the head and lower ex-

[1] Deut. med. Woch., 1901, No. 4, S. 54. (See Case No. 22 in the Summary.)

[2] Magnus-Levy. Hoppe-Seyler's Zeit. f. Phys. Chemie., 1900, Bd. xxx. S. 200.

[3] In the case of the red marrow from the horse's rib, the extract by the ammonium-sulphate method contained a *trace* of albumin coagulating at 75° C. In the case of the human vertebræ by the alcohol method, the application of the nitric and hydrochloric acid tests produced an indefinite haze but no distinct ring, and no coagulation occurred on heating even to boiling temperature.

tremity of the femur and in the ribs, and a thin, red currant-jelly-like mass in the shaft of the femur. We therefore divided the bones into three groups, viz.: (1) vertebræ and ends of femur; (2) ribs; and (3) shaft of femur.

1. *Vertebræ and Ends of Femur.* The neutralized extract by the alcohol method was very opalescent, and could not be cleared by filtration. It was accordingly dialyzed for three days in tap water and for twenty-four hours in distilled water. A slight precipitate separated out, and on filtration a clear solution was obtained. In the dialysates by both methods the following reactions were obtained:

Heat Coagulation. A coagulum was obtained at 70° C., which did not clear upon boiling. On filtering the boiling fluid the filtrate did not show any opacity on cooling. On the addition of one drop of 20 per cent. acetic acid to 30 c.c. of the fluid coagulation occurred at 50° C., but the precipitate did not clear up on boiling, although it appeared to do so to a certain extent, because of the coagulum separating out as flocculi on the surface.

Hydrochloric Acid. A distinct sharp ring was obtained.

Nitric Acid. A similar ring was obtained, which cleared up considerably on heating, reappearing on cooling.

Two volumes of a *saturated solution of common salt and a few drops of 20 per cent. acetic acid.* A precipitate.

A few drops of *acetic acid and potassium ferrocyanide.* A precipitate, not clearing on boiling.

Biuret Test. Violet reaction.

In the opalescent solution by the alcohol method the addition of one drop of 20 per cent. acetic acid produced a precipitate, soluble, however, in excess of the acid.

2. *Ribs.* By the alcohol method an opalescent fluid was obtained, which gave a very distinct precipitate on the addition of acetic acid. The opalescent fluid was dialyzed for several days and filtered.

The clear fluid by both methods gave the following reactions:

Heat Coagulation. Slight haze at 60° C., which did not clear upon boiling.

Nitric and Hydrochloric Acids. A distinct ring was obtained with both of these reagents, which cleared up considerably on boiling, becoming more distinct on cooling. A slight haze was produced by adding 1 in 10 HCl to the fluid.

Acetic Acid, 20 per cent. A few drops of this produced a distinct precipitate, insoluble in excess.

Biuret Test. Slight violet reaction.

The precipitate produced in the undialyzed opalescent solution by the alcohol method on the addition of acetic acid[1] was fused in a silver basin with caustic soda and saltpetre; the resulting mass was dissolved in water, acidified with nitric acid, and mixed at 60° C. with ammonium molybdate solution. No trace of phosphorus was found present.

3. *Shaft of Femur.* The extracts contained only the merest trace of a proteid, and the only reactions which gave anything were the ring tests.

4. *Kidneys.* Slight rings were obtained with the acids, and on boiling the fluid became opalescent.

[1] The amount of precipitate used for this test was quite sufficient for the purpose.

5. *Liver, Spleen, and Muscle.* No trace of proteid was shown by any reaction by either method.

6. *Pericardial Fluid.* Slight rings were obtained with the acids, and on boiling an opalescence was produced. Acetic acid produced no haze. The ammonium-sulphate method was alone employed, as only 45 c.c. of the fluid was examined.

7. *Bile.* No trace of proteid.

8. *Blood.* Fifty-five grammes of clotted blood from the heart was examined. This was extracted with water and the extract examined by both methods. In neither case was the slightest trace of proteid obtained.

Consideration of Results. From an examination of these results it will be seen that in no organ or tissue could the presence of a proteid identical with that found by the same methods in the urine be demonstrated. In the case of the vertebræ and ends of the femur, however, a *proteid giving very similar reactions* was obtained. It will be noticed that the points wherein this differs most from the typical Bence-Jones proteid are the temperature at which it coagulates and the fact that it does not clear up on boiling. It will also be noticed that after the addition of the merest trace of acetic acid the point of heat coagulation was the same as that in a similarly treated extract from urine, but whereas the urinary coagulum cleared up at 90° C., that of the marrow did not do so, even on boiling.

Recent work by K. Spiro,[1] Hammarsten,[2] and others shows, however, that not only does the exact point of heat coagulation vary considerably with the dilution and composition of the fluid, but also that resolution of the coagulum depends to a very large extent on the composition of the fluid in which it is suspended.[3] In view of these facts it is impossible to draw any deductions as to the nature of the body from a consideration of the heat-coagulation point alone, and the mere fact that any proteid whatsoever should have been obtained after the processes to which the original extract was subjected shows in itself *that some unusual proteid existed in the tissues.*

Nor could such a proteid be obtained by employing exactly the same methods from normal red bone-marrow.

The neutralized opalescent solution obtained by the alcohol method gave a copious precipitate on weakly acidifying with acetic acid. This precipitate was soluble in excess of the acid. It was easily soluble in weak alkalies and gave the xanthoproteic and other reactions for proteids. This precipitate was not present in the dialysate obtained by the ammonium-sulphate method. From its reactions it would appear to be either an alkali albumin or a nucleoproteid. To decide this question it was tested for phosphorus, with a negative result. It is probable, therefore, that a certain amount of alkali albumin had been produced out of the alcohol coagula by boiling with dilute ammonia. After the separation of this by neutralization and dialysis, a clear

[1] Hoppe-Seyler's Zeit. f. Phys. Chemie., 1900, Bd. xxx. S. 182.

[2] Pflüger's Arch. f. d. ges. Physiologie, 1878, Bd. xviii. S. 65.

[3] It was noticed, both in the case of the urine and of the vertebral marrow, that on allowing the extract to stand for four weeks in a stoppered bottle a sediment had settled down. This was filtered off, and the clear filtrate failed to give any reaction for proteids. The precipitate was insoluble in weak alkalies. The albumose-like body had evidently undergone a change on standing. There was unfortunately not sufficient material to reinvestigate this fact.

solution giving exactly the same reactions as the ammonium-sulphate extracts was obtained.

The extract obtained from the red currant-jelly-like marrow found in the shaft of the femur contained only the minutest trace of proteid.

The only other tissue from which a proteid-containing extract could be obtained was the kidney, and here only in the minutest trace. The pericardial fluid gave rather clearer reactions than the kidneys, a result in accordance with Ellinger's observations.

Conclusions Regarding the Seat of Production of Bence-Jones Proteid (Hutchison and Macleod).

The invariable, or almost invariable, occurrence of bone-marrow disease in cases of Bence-Jones albumosuria would seem to point out the seat of production of this unusual proteid; but yet, when one considers the enormous quantities excreted daily—15 grammes in the above case—it is at first sight difficult to conceive how such a source can be possible. Magnus-Levy calculates that the whole mass of the diseased red marrow could not contain more than 100 grammes of proteid, and argues that in his case,[1] at least, where the daily excretion in the urine often attained 36 grammes, its derivation from the bones was impossible. In support of this contention he points out that in those cases where the total urinary nitrogen was estimated it was found that nearly 40 per cent. of this was excreted as Bence-Jones proteid. In a case investigated by Seegelken,[2] however, only about 10 per cent. of the total nitrogen was so excreted. From a consideration of these facts Magnus-Levy supposes that the bone-marrow cannot be the seat of its production, but that it represents a non-assimilated digestive proteid. This want of assimilation he ascribes to the absence of some influence which the bone-marrow normally exercises on the metabolism of proteid, but which is wanting when the marrow is diseased.

In the above case, however, the results point to the bone-marrow as the seat of production, and the absence of any proteid in the extract from nearly all the other tissues and organs would tend to disprove Magnus-Levy's theory, for were this correct we should expect to find at least some of the unusual proteid present in those organs (muscles, liver, etc.) where proteid metabolism is most active.[3]

The body was not found in the blood, and this was probably due to the fact that only a limited quantity was procurable for examination. Its presence in the kidneys was to be expected, since a certain amount of urine must still have been present in the tubules.

Remarks on Multiple Myeloma in General.

Multiple myeloma may be defined as a diffuse new-growth primarily involving the bone-marrow, especially that of the vertebræ, ribs, and sternum,[4] and affecting males as or more often than females, and

[1] Case No. 11 in the Summary.

[2] Deut. Arch. f. klin. Med., Bd. lviii. S. 276. (See Case No. 6 in the Summary.)

[3] It is certainly difficult to conceive how so much proteid could be derived from so small a source; but still, when we take into account other metabolic processes in the body, *e. g.*, the occurrence of 30 grammes of urea in the urine and of only 2 grammes in the whole body, the result does not seem so surprising.

[4] In the present case, however, and in some other cases (see Nos. 20 and 24 in the Summary), the bones of the limbs were likewise much affected. Doubtless if the whole skeleton had been

chiefly those past middle age. The disease nearly always remains limited to the osseous system, though by direct extension it may form localized outgrowths projecting from the bones. Owing to absorption of the hard, osseous tissue the bones become softened or friable, and are easily broken. The vertebral column and sternum are sometimes much bent, and the spinal cord may be affected by pressure, due to the curvature of the spinal column or to new-growths bulging into the spinal canal. Owing to the destruction of bone-marrow, the formation of blood is impaired, and anæmia and progressive cachexia occur, doubtless in some cases favored by the circulation of a toxic proteid. I cannot help drawing an analogy between the bone disease, myelomatosis (*i. e.*, multiple myeloma), on the one hand, and the skin disease, mycosis fungoides, on the other. In both cases the etiology, as well as the true nature of the new-growth, is obscure. For both diseases an infection theory has been propounded, making the new-growths allied to the class of infective granulomata; but at present the arguments in favor of any such infection theory are far from convincing. In myelomatosis there is a primary diffuse infiltration of the bone-marrow of a great part of the skeleton, followed in some cases by the formation of definite localized tumors growing from the bones; while mycosis fungoides usually commences as a diffuse infiltration of the skin (premycotic stage), and the localized tumors, which give the disease its name, begin to sprout out later on. This analogy may perhaps turn out to be a very superficial one, but in the present uncertainty regarding both diseases it is worthy of mention. In one important point the analogy is imperfect. The point is that while mycosis fungoides seems to be a single definite disease (morbid entity), different kinds of neoplasm have apparently been included under the heading "multiple myeloma."

Multiple myeloma is a term which has been employed to include various diffuse new-growths arising in the bone-marrow (*i. e.*, myelogenic), and not giving rise to definitely metastatic growths in other tissues.[1] After post-mortem investigations various names have been employed according to the histological features (and individual interpretations by observers) of the neoplasms, and particularly of the cell elements of which the tumors are formed. The tumors have been regarded as simple overgrowth of the cell elements of the bone-marrow, or as myelogenic sarcoma, endothelioma, perithelioma, plasmoma, etc.

In my first case[2] of "multiple myeloma" I supposed the tumor

examined the long bones would have been found affected in some of the cases in which by the clinical symptoms the bone disease was supposed to be limited to the ribs, sternum, and vertebral column.

[1] See later on in regard to the lymphatic glands becoming affected in some cases.

[2] General Lymphadenomatosis of Bones, One Form of Multiple Myeloma. Transactions of the Pathological Society of London, 1897, loc. cit. The patient is referred to later on as E. P.

formation to be an example of "general lymphadenomatosis of bones." Recent re-examination of old sections (stained by the ordinary methods) from the bone-marrow growth and from the affected glands has confirmed the view and made it practically certain that the growth in the glands is identical with that in the bones. The cells of which the growth consists resemble lymphocytes, except that very many of them have more protoplasm than ordinary small lymphocytes have. The larger cells are rounded, oval, or polyhedral, and the nucleus is often placed excentrically. Part of the sternum was fortunately preserved in glycerin and formalin in the Museum of the Royal College of Surgeons, and thus I have lately had an opportunity of examining sections of the sternal portion of the growth stained by Ehrlich's triacid and by Mann's eosin-methyl-blue combinations. These stains show that hardly any of the cells contain granules. In fact, only one or two coarsely granular eosinophile cells were seen in looking through the sections, and these were probably not tumor cells. The tumor may, therefore, be said to consist of non-granular, lymphocyte-like cells. The majority of these cells have more protoplasm than ordinary small lymphocytes have. It must be remembered, however, that in lymphatic leucocythæmia the growths in the various organs may consist largely of cells which, though they are described as lymphocyte-like, have much more protoplasm than do the small lymphocytes of normal blood. Every intermediate form between the cells with much protoplasm and those with very little protoplasm can be found in the growths of lymphatic leucocythæmia, and so they can be in the myelogenic growth from the patient E. P. Moreover, in normal lymphatic glandular tissue many of the cells of the "germinating centres" have more protoplasm than the small lymphocytes, further from these centres, and in the circulating blood. I feel justified, therefore, in saying that in the case of E. P. the cells of the myelogenic growth were lymphocyte-like, if not actually of the lymphocyte kind. Dr. J. M. H. MacLeod has kindly examined the growth by special staining for plasma cells, and thinks that the cells of which the growth is composed have a greater resemblance to lymphocytes than to the typical plasma cells of the granulomata.[1]

Two types, if not more, of "multiple myeloma" are to be distinguished: (1) a growth, as in the patient, J. T., in which the bone-marrow only is involved; (2) a growth in which nearly all the cells resemble small or large lymphocytes, and are possibly derived from the non-granular predecessors of the myelocytes;[2] in this second type

[1] I may add that in part of the growth in the sternum many of the tumor cells have undergone some kind of a degenerative change, owing to which, by Mann's eosinmethyl-blue combination, the nucleus and the rest of the cell are deeply stained by the eosin.

[2] A. Pappenheim. Virchow's Archiv, 1902, vol. clxix. p. 381. According to his views, the *large lymphocyte* type of cell, which in the lymphatic glands gives rise to the ordinary small

of "multiple myeloma" lymphatic glands as well as bone-marrow may probably be affected. The second type of multiple myeloma would include cases described as myelogenic lymphosarcoma, myelogenic lymphadenoma, and myelogenic pseudoleukæmia (using the German term "leukæmia" in the limited sense of "lymphocythæmia)." Intermediate cases between these two types ("mixed forms") probably also occur.

If the views which I have suggested in this paper be correct, it follows that the *whole class of leukæmias and pseudoleukæmias* (using the German terms for convenience) can be divided into at least the following six types, independently of intermediate forms:

(*a*) A new-growth of lymphocyte-like cells originating in the bone-marrow and not overflowing into the circulating blood. Myelogenic pseudoleukæmia (using leukæmia in the sense of lymphocythæmia), myelogenic lymphosarcoma, lymphadenomatosis of bones, multiple myeloma (myelomatosis) of the lymphatic type.

(*b*) Similar to the preceding, but the lymphocyte-like cells overflow into the blood stream. Myelogenic lymphocythæmia. I do not know of any cases illustrating this type excepting cases of "acute leukæmia." Those of A. Dennig (*Münchener med. Wochenschrift*, 1901, No. 4, page 140) and C. H. Melland (*Medical Chronicle*, September, 1902, page 372), for instance, were examples of acute lymphocythæmia in which *post-mortem* practically no change was discovered in leucocyte-forming tissues other than the bone-marrow.

(*c*) A new-growth formed in large part of lymphocyte-like cells originating in the lymph glands or the lymphadenoid tissues generally, and not to any great extent overflowing into the circulating blood. Lymphatic or splenic lymphadenoma or pseudoleukæmia (using leukæmia in the sense of lymphocythæmia), Hodgkin's disease. In the more chronic and fibrous varieties of this type the microscopic appearances differ, of course, considerably from those in acute cases.

(*d*) Similar to the preceding, but the lymphocyte-like cells invade the blood stream. Lymphatic or splenic lymphocythæmia.

(*e*) A new-growth in the bone-marrow of cells derived from the myelocytes not invading the circulating blood. Myelogenic pseudoleukæmia (using leukæmia in the sense of myelogenic or splenomedullary leucocythæmia). To cases of this type and to "mixed cases" partaking of this type the term *multiple myeloma* (*myelomatosis*) might perhaps be limited.

(*f*) A new-growth characterized by its myelocyte-like cells overflowing or being drawn out into the circulating blood and by Bence-Jones

lymphocytes of the blood, in the bone-marrow gives rise to the myelocytes and thus indirectly to the polymorphonuclear leucocytes also.

albumosuria, not occurring as it sometimes does in the preceding type. Myelogenic or splenomedullary leukæmia (leucocythæmia).

According to this scheme, one must regard the excess of white corpuscles in the blood in all kinds of leukæmia as due to an inroad of tumor cells from a *hyperplasia-like* tumor formation in the leucocyte-producing tissues of the body,[1] all forms of leucocytes (including lymphocytosis) being merely expressions of some reaction in the tissues in question. A leucocytosis is, therefore, strictly speaking, never an early stage of leukæmia (leucocythæmia); yet a true leucocytosis from any cause may perhaps sometimes be followed by true leukæmia in so far as a reactive growth in leucocyte-producing tissues (of which reactive growth the leucocytosis is the expression) may be supposed to give a start to the kind of tumor formation of which leukæmia is the expression, just as chronic irritation of the skin sometimes acts as the exciting cause of epithelioma.

The Bence-Jones Proteid in the Urine of Multiple Myeloma Cases.

Drs. Hutchison and Macleod concluded that in the present case the bone-marrow was probably the seat of production of the proteid in question, and that this proteid is not a non-assimilated digestive proteid, as suggested by Magnus-Levy.[2] As confirming their views, I found that a considerable alteration of diet maintained during two days had no effect in altering the quantity of proteid excreted in the urine. Moreover, Dr. Bradshaw in his case found that meals had little or no influence on the excretion of the proteid in the urine. He found that as much was excreted by night as by day,[3] when the patient was taking meals during the daytime only; and he considered the rate of excretion to be "pretty constant throughout the twenty-four hours."

It seems that when it is free in the blood the Bence-Jones proteid appears in the urine,[4] as hæmoglobin does whenever owing to various causes sudden unusual hæmolysis occurs. The fact, therefore, that the quantity excreted in the urine is little influenced by meals and by change of diet speaks strongly against the correctness of Magnus-Levy's views. It is possible that the cells of the new-growth in the bone may produce digestive enzymes, by the action of which on the albuminous constituents of the blood serum the Bence-Jones proteid is steadily and continually manufactured; then from the circulating blood it would pass through the renal filter with the urine, like hæmo-

[1] Pappenheim's various writings, loc. cit., etc. [2] Loc. cit.

[3] See the tables at the end of Dr. Bradshaw's first paper. Med.-Chir. Trans., 1898, vol. lxxxi. pp. 270, 271.

[4] Sir Lauder Brunton has kindly directed my attention to certain experiments of B. J. Stokvis, published in the Maandblad der Sectie voor Natuurwetenschappen, 1872, No. 6. The latter found that Bence-Jones proteid, when a not very concentrated solution was injected into the rectum of a dog, was excreted unchanged in the urine.

globin, even in the absence of any kidney disease. As already mentioned, there may be some connection between the excretion of the proteid in the urine and the formation of granules and globules in the cells of the new-growth in the present case.

Diagnosis of Multiple Myeloma (Myelomatosis), with and without Bence-Jones Proteid in the Urine.

There is still much uncertainty as to the nature of multiple myeloma (multiples myelom), a term first employed by J. von Rustizky,[1] who regarded the growth as formed by a simple hypertrophy of bone-marrow. As already mentioned, however, the tumors from different cases do not all resemble each other in their histological features, though they possess certain characters in common. The growth is generally so diffuse in its distribution that it is impossible to determine that any one part represents a primary focus where the neoplasm may be supposed to have commenced. It does not invade other tissues by metastasis through the blood channels as sarcoma does, though in some cases the lymphatic glands have been involved (cf. Case 3 in the Summary at the end).

Owing to the softening and fragility of bones, the pains and the progressive kyphosis caused by the disease, the diagnosis is from:

(*a*) *Osteomalacia.*

(*b*) *Muscular rheumatism, lumbago, sciatica, etc.*

(*c*) *Spondylitis deformans.*

(*d*) *Caries of the spinal column.*

(*e*) *Invasion of the vertebral column and other bones by secondary malignant tumors.* Owing to the progressive anæmia and cachexia, one may think of:

(*f*) *Pernicious anæmia or other diseases associated with progressive cachexia.* Owing to the possibility of confusing Bence-Jones proteid in the urine with albumin, the cases of multiple myeloma in which this body is present in the urine (*i. e.*, the cases of "myelopathic albumosuria" of Bradshaw, "Kahler's disease") may be at-first mistaken for:

(*g*) *Nephritis.*

(*a*) *Osteomalacia.* From the typical osteomalacia of women multiple myeloma differs in the following respects. The former attacks women during the childbearing period of life. It affects chiefly the bones of the pelvis and lower extremities. It gives rise to great deformity by the bending of the bones, but more rarely to "spontaneous" fractures. Multiple myeloma attacks men as often as women, or more often, and chiefly those in the second half of life. Clinically, it appears specially to affect the vertebral column, ribs, and sternum, though the bones

[1] Multiples Myelom. Deutsche Zeitschrift f. Chirurgie, Leipzig, 1873, vol. iii. p. 162.

of the extremities have certainly been involved in some cases, as they were in my present case. It is likely to cause fractures of the ribs and deformity by bending of the vertebral column and sternum; in one case (Case No. 20 in the Summary) "spontaneous" fracture of one femur occurred, and in another (Case No. 24 in the Summary) "spontaneous" fractures of both femora are recorded; yet it does not give rise to the characteristic deformities of osteomalacia resulting from yielding of the pelvis and bending of the bones of the lower extremities. It is possible, however, that there may be true cases of osteomalacia in males and in elderly females in which the bones of the vertebral column and trunk are specially affected.

(*b*) *Muscular Rheumatism, Lumbago, Sciatica, etc.* Several cases of multiple myeloma have, at least during part of the disease, been given such headings. The occurrence of markedly bilateral thoracic, abdominal, or lumbago-like pains may first direct attention to the possibility of disease of the spinal column. In my first case of multiple myeloma,[1] pain on both sides of the abdomen, together with the presence of an increasing kyphosis, pointed to grave disease of the spinal vertebræ. Sometimes examination of the bones by Röntgen rays may prove of service (*vide* Case No. 13 in the Summary at the end). If grave rheumatoid or rheumatic arthritis is a complication, as in my present case, it obviously becomes difficult to distinguish pains and paræsthesiæ due to the arthritis from those due to the multiple myeloma.

(*c*) *Spondylitis Deformans.* This affection of the vertebral column may produce a similar kyphosis to that which in several cases has been caused by multiple myeloma. When, however, the kyphosis is due to spondylitis deformans,[2] the spinal rigidity in the cervical region is probably more pronounced than in cases of multiple myeloma, while the patient is likely to be less anæmic and cachectic.

(*d*) *Caries of the Spinal Column.* The progressive bending of the vertebral column seen in multiple myeloma might be confused with tuberculous caries, that is, with those rather rare cases occurring in middle or old age and giving rise to progressive curvature. On the other hand, the curvature due to myeloma is somewhat less likely to be distinctly "angular" than that due to tuberculosis, and in the latter disease the ribs are not likely to be in any way affected simultaneously

[1] Trans. Path. Soc., loc. cit.

[2] Cases in which the vertebral column only is affected (von Bechterew's type) may be distinguished from those in which the extremities, especially the hip-joints, are likewise affected (Strümpell's type, Pierre Marie's "spondylose rhizomélique"). Such cases of chronic ossifying arthritis may progress to universal bony ankylosis. (See the summary of cases by Dr. Joseph Griffiths in the Journal of Pathology and Bacteriology, December, 1896, and March and June, 1897. Much has been written on the subject during recent years in France and Germany.) Of course, when ankylosis of the joints of the extremities has occurred, a case could hardly be mistaken for one of multiple myeloma, but even at the commencement of the disease such a mistake is very unlikely to be made.

with the spinal column. The sternum has sometimes become excessively bent in multiple myeloma. The presence of tuberculosis in the lungs might help to clear up the diagnosis.

(*e*) *Invasion of the Vertebral Column and Other Bones by Secondary Malignant Tumors.* Secondary localized malignant tumors may give rise to a progressive curvature of the vertebral column. The vertebral column and ribs may likewise be diffusely infiltrated by metastatic carcinoma, but all such metastatic growths are more likely to cause distinct swellings[1] or give rise to local signs of their presence. Evidence as to a primary malignant growth existing or having been removed from some other part of the body would facilitate the diagnosis, and in localized tumors, as well as in tuberculous caries, help might be obtainable from the Röntgen rays.

(*f*) *Pernicious Anæmia, etc.* Anæmia and progressive wasting and feebleness may be marked features of myelomatosis, at all events in the later stages, when the blood-forming functions of the bone-marrow are greatly impaired by the diffuse tumor formation. The pains and other signs of bone disease, such as progressive kyphosis, when these are well marked, will help to distinguish cases of multiple myeloma from pernicious anæmia and forms of progressive cachexia dependent on visceral cancer, etc.

(*g*) *Nephritis.* The Bence-Jones proteid when present in the urine may be confused with albumin, and the case may be regarded as one of nephritis. This is especially likely to occur if the urine on the first occasion is examined very hurriedly (for example, by heating it without boiling it, or by merely adding picric acid or nitric acid in the cold), and, owing to the copious precipitate of supposed ordinary albumin, it is subsequently examined every day by Esbach's tube. This actually occurred in the present case,[2] where the general "puffy" look of the patient seemed to correspond to the finding of albuminuria. Moreover, in this case, as in some other cases, hyaline casts were found in the urine.[3] Afterward the testing of the urine by the ordinary methods, instead of by Esbach's solution, led to the detection of the special proteid it contained.

I need not repeat what has already been said in regard to the tests

[1] In some cases, however, of multiple myeloma, with or without Bence-Jones proteid in the urine, localized tumors connected with the bones could be seen or felt during life. (See Cases 14, 19, 27, and 33 in the Summary at the end.)

[2] The remarkable *constancy in the amount* and the *large quantity* of the proteid in the urine ought perhaps to have caused some surprise.

[3] The necropsy, it should be remembered, showed the presence of actual interstitial fibrotic changes in the kidneys. In Senator's case (Case No. 7 in the Summary) casts and albumin were present in the urine, and the kidneys were found to be somewhat diseased at the post-mortem examination. In d'Allocco's case (No. 15) there was likewise nephritis; in Ellinger's (No. 9) the urine contained a few hyaline casts; and in Çonti's (No. 27) during the last weeks of life the urine contained albumin and hyaline and granular casts.

to be employed for distinguishing Bence-Jones proteid in the urine from albumin and from certain albumoses.[1] In the albumosuria occasionally met with in cases of intestinal ulceration and febrile disorders the quantity of proteid in the urine is generally far less than in Bence-Jones albumosuria. In such a case as that recorded by Dr. R. Hutchison,[2] where the proteid, though precipitated at as low a temperature as 58° C., was not, even partially, redissolved on heating to the boiling point, the chances of wrongly regarding the precipitate as one of ordinary urinary albumin must be still greater than they were in the present case, where the greater part of the precipitate redissolved on boiling the urine.

I now come to the *diagnostic value of finding Bence-Jones proteid in the urine.* From a study of cases the following conclusions must be arrived at: 1. Undoubtedly a considerable number of cases of multiple myeloma have occurred in which the Bence-Jones proteid has not been detected. In some of them the urine may have been examined at a stage of the disease prior to the commencement of the "Bence-Jones albumosuria."[3] In other cases the urine may possibly have been insufficiently examined. Still there remain sufficient cases to enable one to affirm with almost absolute certainty that "multiple myeloma" may occur without giving rise to the presence of Bence-Jones proteid in the urine. It must be remembered, however, that different types of tumor have been included under the heading "multiple myeloma," but I will not repeat here the conclusions which I have already mentioned in an earlier part of the present paper. 2. Metastatic tumors affecting the skeleton, however extensively the bone-marrow be infiltrated, have never yet been known to cause "Bence-Jones albumosuria." 3. The presence of Bence-Jones proteid in the urine is practically invariably of fatal significance, and nearly always, if not always, indicates that the patient is suffering from "multiple myeloma." 4. One or two published cases in which Bence-Jones proteid was present in the urine seem, however, to have been exceptions to the rule in that they were supposed not to be instances of multiple myeloma. Moreover, the experiments of Dr. G. Zuelzer,[4] should they be confirmed, would make the existence of such exceptions more prob-

[1] I do not think that Dr. L. N. Boston's caustic soda and lead acetate test (A Rapid Reaction for Bence-Jones Albumose, THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, October, 1902, p. 567) is likely to be of much service in this connection.

[2] Case No. 30 in the Summary of Cases at the end.

[3] In the case of Stokvis and Kühne (Case No 2 in the Summary) the Bence-Jones proteid is said to have appeared late in the disease, and could not be found three months before the patient's death. Conti says that during the last weeks of life in his case (No. 27 in the Summary) the urine contained albumin, but no Bence-Jones proteid.

[4] Ueber experimentelle Bence-Jones'sche Albumosurie. Berliner klinische Wochenschrift, 1900, No. 40, p. 894. See also Campbell-Horsfall's account of *temporary* Bence-Jones albumosuria in a patient after severe injury to a leg, Lancet, London, April 25, 1903.

able. He rendered a dog anæmic by giving it pyrodin by the mouth. On the eighth day from the commencement of the experiment a substance was detected in the urine giving the typical reactions for Bence-Jones proteid, and no albumin was present. The pure Bence-Jones albumosuria lasted four days, and then albuminuria occurred and the amount of the Bence-Jones proteid diminished. It would be interesting to know what changes occurred in the bone-marrow of this animal.

Taking all the data that I can obtain into consideration, it seems to me quite possible: 1. That Bence-Jones albumosuria is always the result of disease of the bone-marrow. 2. That it is due to an abnormal metabolic or degenerative process in the myelocytes or in tumor cells derived from the myelocytes or their predecessors. 3. That the reason why it is generally, though not always,[1] associated with myelogenic tumor formation is that the tumor cells derived from bone-marrow cells, however much they may morphologically resemble true bone-marrow cells, are more prone to abnormality (including unusual degenerative changes) than real myelocytes are. 4. That non-myelogenic tumor cells are not affected in the same way, and therefore metastatic tumors in the bone-marrow do not give rise to Bence-Jones albumosuria.

I shall now give references to all the reported cases in which Bence-Jones proteid has been detected in the urine,[2] whether the presence of bone disease was verified or not. I shall also refer to some doubtful cases in which the reactions of the proteid in the urine were not quite characteristic for Bence-Jones proteid, and shall mention certain supposed cases obviously incorrectly included in previous summaries on the subject. For full details, the original papers, to which references are given under each case, must be consulted, but for short abstracts of the earlier cases Dr. C. E. Simon's paper in THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES for June, 1902, may be consulted. Among doubtful cases are those, such as that reported by R. Hutchison (Case No. 30), in which, though a copious precipitate occurs on slightly heating the urine, yet this precipitate is not to any extent dissolved by further heating. In this connection it must be remembered that the

[1] An analogy between Bence-Jones albumosuria and melanuria may be made. The presence of melanin and melanogen in the urine is best known in cases of melanotic tumor, but has been noted likewise in wasting diseases. Melanotic tumors, however, are not always associated with the excretion of melanin or melanogen in the urine; yet, as Dr. A. E. Garrod points out (St. Bartholomew's Hospital Reports, vol. xxxviii. p. 25), melanuria has been undervalued for diagnostic purposes because other conditions in which the urine blackens on exposure to air have been confounded with true melanuria.

[2] I shall not here refer to the cases of "multiple myeloma without albumosuria," several of which are quoted in my paper in vol. xlviii. of the Transactions of the Pathological Society of London (loc. cit.). On the whole subject of multiple myeloma, the important writings of F. W. Zahn, Deut. Zeitschr. f. Chirurgie, 1885, vol. xxii. p. 1; Hammer, Virchow's Archiv, vol. cxxxvii. p. 280; Markwald, ibid., vol. cxli. p. 128; R. Paltauf, Ergebnisse der allg. Pathologie, edited by Lubarsch and Ostertag, 1896, vol. i. pp. 676–679; K. Winkler, Virchow's Archiv, vol. clxi. p. 252; E. Wieland, ibid., vol. clxvi. p. 108; and M. Borst, Die Lehre von den Geschwülsten, 1902, vol. i. pp. 492–494, may be consulted.

experiments of Hammarsten,[1] K. Spiro,[2] Magnus-Levy,[3] and others in regard to various proteids show that the point of heat coagulation varies with the dilution and composition of the fluid, and the resolution of the coagulum depends likewise, to a great extent, on the composition of the fluid in which it is suspended. Among cases which should not be included in the Summary are those of albumosuria (other than "Bence-Jones albumosuria") in which no precipitate occurs on merely heating the urine (presumably acid in reaction), but in which the addition of nitric acid to the cold urine gives rise to a precipitate, which dissolves on heating and reappears on cooling.[4]

Summary of Cases of Bence-Jones Albumosuria.

No. 1. *The Case of Watson, Macintyre, and Bence-Jones, of London.* (H. Bence-Jones, "On a New Substance Occurring in the Urine of a Patient with Mollities Ossium," *Phil. Trans. Royal Society of London*, 1848, part i., p. 55. W. Macintyre, "Case of Mollities and Fragilitas Ossium," *Med.-Chir. Trans.*, London, 1850, vol. xxxiii., p. 211. J. Dalrymple, "On the Microscopic Character of Mollities Ossium," *Dublin Quarterly Jour. Med. Sci*, vol. ii., p. 85, 1846.)

No. 2. *The Case of Doornik, Stokvis, and Kühne.* (W. Kühne, "Ueber Hemialbumose im Harne," *Zeitschrift f. Biologie*, 1883, vol. xix., p. 209.)

No. 3. *The Case of Kahler and Huppert.* (O. Kahler, "Zur Symptomatologie des Multiplen Myeloms: Beobachtung von Albumosurie," *Prager medicinische Wochenschrift*, 1889, vol. xiv., p. 33. H. Huppert, "Ein Fall von Albumosurie," *ibid.*, p. 35.)

No. 4. *The Case of Stokvis, Ribbink, and Zeehuisen.* (B. J. Stokvis, *Nederl. Tijdschrift voor Geneesk.*, 1891, vol. ii., p. 36. H. C. G. Ribbink's *Dissertation*, Amsterdam, 1892. H. Zeehuisen, *Nederl. Tijdschrift voor Geneesk.*, 1893, vol. i., p. 829. See also abstracts in Maly's *Jahresbericht f. Thier. Chem.*, vol. xxi., p. 412; vol. xxii., p. 525; vol. xxiii., p. 577.)

No. 5. *The Case of Raschkes.* (A. Raschkes, "Ein Fall von seniler Osteomalacie mit Albumosurie," *Prager med. Wochenschrift*, December 20, 1894, No. 51, p. 649.)

No. 6. *The Case of Professor Stintzing, of Jena.* (Seegelken, "Ueber Multiples Myelom und Stoffwechsel Untersuchungen bei derselben," *Deut. Archiv f. klin Med.*, 1897, vol. lviii., p. 276. M. Matthes, "Ueber Eiweisskörper im Urine bei Osteomalacie," *Ver-*

[1] Loc. cit. [2] Loc. cit. [3] Loc. cit.

[4] A typical example of this is recorded by Hougounenq, Lyon Médical, January 20, 1901, vol. xcvi. See, also, E. Vidal's case, Comptes rendus de la Société de Biologie, October 29, 1898, p. 991, in a woman, aged twenty-four years, suffering from tuberculous disease of the right shoulder. Dr. J. A. Blair's Case of Albumosuria, British Medical Journal, September 14, 1901, p. 713, is doubtless of the same kind. He states that the urine "gave no perceptible precipitate on simple heating without acid," but that on adding nitric acid to the cold urine a precipitate occurred which was dissolved on heating and reappeared on cooling. This kind of albumosuria is doubtless much less rare than the Bence-Jones albumosuria, and is probably sometimes altogether overlooked owing to the fact that the boiling test for albumin is more often employed than the nitric acid (cold) test. It must be remembered, however, that in true Bence-Jones albumosuria the urine, if alkaline, should likewise not be expected to give any precipitate on heating until it has been rendered slightly acid, *e. g.*, by the addition of acetic acid.

handl. des XIV. Congresses f. innere Medicin, 1896, p. 476. R. Neumeister, *Lehrbuch der Physiol. Chemie*, 1897, second edition, p. 804.)

No. 7. *Senator's Case.* (H. Rosin, "Ueber einen eigenartigen Eiweisskörper in Harne und seine diagnostische Bedeutung," *Berliner klin. Wochenschrift*, 1897, p. 1044. H. Senator, "Asthenische Lähmung, Albumosurie und Multiple Myelome," *Berliner klin. Wochenschrift*, 1899, p. 161. The urine has likewise been studied in Süssmann's *Dissertation*, Leipzig, 1897.)

No. 8. *Bozzolo's Case.* (Camillo Bozzolo, "Sulla Malattia di Kahler," in the *Transactions of the Eighth Medical Congress*, Naples, October, 1897; and in *La Clinica Medica Italiana*, Milan, January, 1898, p. 1; referred to also in *Centralblatt f. die med. Wissenschaft*, 1898, vol. xxxvi., p. 572.)

No. 9. *The Case of Lichtheim and Ellinger.* (A. Ellinger, "Das Vorkommen des Bence-Jones'schen Körpers im Harn bei Tumoren des Knochenmarks und seine diagnostische Bedeutung," *Deut. Archiv f. klin. Medicin*, 1899, vol. lxii., p. 255.)

No. 10. *Bradshaw's Case.* (T. R. Bradshaw, "A Case of Albumosuria in which the Albumose was Spontaneously Precipitated," *Med.-Chir. Trans.*, London, 1898, vol. lxxxi., p. 259. Bradshaw and Warrington, "The Morbid Anatomy and Pathology of Dr. Bradshaw's Case of Myelopathic Albumosuria," *ibid.*, 1899, vol. lxxxii., p. 251. Bradshaw, *Transactions of the Pathological Society*, London, 1900, vol. li., p. 140. Bradshaw, "Myelopathic Albumosuria," *British Medical Journal*, November 3, 1900, p. 1304. Bradshaw, "Myelopathic Albumosuria," *Lancet*, October 4, 1902, p. 929.)

No. 11. *Case of Naunyn and Magnus-Levy.* (Naunyn, "Ein Fall von Albumosurie," *Deutsche med. Wochenschrift*, 1898, *Vereins-Beilage*, p. 217. A. Magnus-Levy, "Ueber den Bence-Jones'schen Eiweisskörper," Hoppe-Seyler's *Zeitschrift f. Phys. Chemie*, 1900, vol. xxx., p. 200.)

No. 12. *The First Case of Dr. Fitz.* (R. H. Fitz, "The Significance of Albumosuria in Medical Practice," THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1898, vol. cxvi., p. 30.)

No. 13. *The Second Case of Dr. Fitz.* (R. H. Fitz, *loc. cit.*, p. 42. J. H. Wright, "A Case of Multiple Myeloma," *Johns Hopkins Hospital Reports*, 1900, vol. ix., p. 359; also *Journal of the Boston Society of Medical Sciences*, 1900, vol. iv., p. 195.)

No. 14. *The Case of Buchschtab and Schaposchnikow.* (L. Buchschtab and B. Schaposchnikow, "Diffuses Myelom der Rumpfknochen mit einer Typischen Albumosurie," abstract in German in *St. Petersburger med. Wochenschrift*, 1899, from *Russ. Arch. Pat., Klin., Med., Bact.*, vol.vii.; also abstract in *Centralblatt f. Allg. Pathologie*, 1899, vol. x., p. 589.)

No. 15. *D'Allocco's Case.* (D'Allocco, "Sulla Malattia di Kahler," at the Tenth Medical Congress, Rome, October, 1899, *Arch. Ital. di Medicina Interna*, 1900, vol. iii., fasc. Nos. 1 and 2; referred to also by U. Flora in his article, "Sulla Malattia di Kahler," *Rivista Critica di Clinica Medica*, Florence, 1900, Nos. 46 and 47.)

No. 16. *The Case of Lichtheim and Askanazy.* (S. Askanazy, "Ueber die Diagnostische Bedeutung der Ausscheidung des Bence-Jones'schen Körpers durch den Harne," *Deutsche Archiv f. klin. Medicin*, 1900, vol. lxviii., p. 34.)

No. 17. *Latzko and Sternberg's Case.* (M. Sternberg, in "Vege-

tationsstörungen und Systemerkrankungen der Knochen," *Nothnagel's Spec. Path. u. Therapie*, 1899, vol. vii., part ii., division 2, p. 57.)

No. 18. *Barr's Case.* (J. Barr, "Case of Myelopathic Albumosuria," *Liverpool Med.-Chir. Journal*, March, 1901, p. 23.)

The patient was a man, aged fifty-three years. There were pains in the spinal column, a swelling in the manubrium sterni, etc. The bone disease seemed to be of progressive character. Barr found the quantity of urine usually between forty and sixty ounces daily, and it rarely contained less than twelve per mille of the Bence-Jones proteid. I understand that no post-mortem examinaton was made. In connection with my own recent case it is interesting to note that Dr. Barr's patient presented undoubted signs of cardiac valvular disease.

No. 19. *The Case of a French Physician.* (T. R. Bradshaw, "On the Evolution of Myelopathic Albumosuria," *British Medical Journal*, July 13, 1901, p. 75. Obituary notice on Dr. P. F. Colrat, *British Medical Journal*, September 7, 1901.)

The patient was a well-known French physician of Lyons, aged fifty-five years. The Bence-Jones proteid first appeared in the urine in December, 1899; the amount was at first very little, but it gradually increased and ultimately reached a maximum proportion of ten per mille. Death occurred from pneumonia in August, 1901. In this case Bradshaw (*Lancet*, October 4, 1902, p. 931) says he observed a semi-fluctuating swelling of about the size of a large hen's egg in connection with one of the ribs.

No. 20. *The Case of Iglehart, Hamburger, and Simon.* (L. P. Hamburger, "Two Examples of Bence-Jones Albumosuria associated with Multiple Myeloma," *Johns Hopkins Hospital Bulletin*, February, 1901, p. 38. C. E. Simon, "Observations on the Nature of the Bence-Jones Albumin," THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, June, 1902, p. 939.)

The patient was a lady, aged forty-nine years, whose first symptom was a sharp pain over one of the ribs, in August, 1900. The examination of the urine led to the clinical diagnosis of multiple myeloma. Spontaneous fracture of the left femur occurred a few days before the patient's death in August, 1901. No necropsy was made.

No. 21. *The Case of Osler, Hamburger, and MacCallum.* (Hamburger, *loc. cit.* W. G. MacCallum, "A Case of Multiple Myeloma," *Journal of Experimental Medicine*, 1901, vol. vi., p. 53.)

The patient was a colored woman, aged fifty years, admitted to the Johns Hopkins Hospital in October, 1900. The urine contained Bence-Jones proteid, and the clinical diagnosis of multiple myeloma was confirmed by the necropsy findings recorded by MacCallum.

No. 22. *Kalischer's Case.* (S. Kalischer, "Ein Fall von Ausscheidung des Bence-Jones'schen Eiweisskörper," *Deutsche med. Wochenschrift*, 1901, No. 4, p. 54.)

The patient was a woman, aged sixty-seven years. In this case a spontaneous precipitate (supposed to be of the Bence-Jones proteid) was observed in the urine on allowing it to stand for a long time. After death, examination of the ribs showed a tumor-like hyperplasia of the bone-marrow. Professor Löwy failed to detect the presence of Bence-Jones proteid in the bone-marrow from the ribs and humerus. It may be noted that one of the patient's daughters had died, at the age of thirty years, of "pernicious anæmia."

No. 23. *Rostoski's Case.* (O. Rostoski, "Albumosurie und Peptonurie," at the meeting of the Phys.-Med. Ges. zu Würzburg, June 13, 1901, reported in the *Sitzungs-Berichte*, 1901, Nos. 2 and 3; abstract in *Münchener med. Wochenschrift*, July 2, 1901.)

Rostoski gives no details of this case, but from the results of his examination of the urine says that he inclines to the view of Magnus-Levy that the Bence-Jones proteid (at least, what he found in his case) is not to be classed as an albumose. He has likewise recorded certain investigations regarding the Bence-Jones body in his paper, "Zur Kenntnis der Präcipitine" (Würzburg, 1902).

No. 24. *The Case of Jochmann and Schumm.* (G. Jochmann and O. Schumm, "Typische Albumosurie bei echter Osteomalacie," *Münchener med. Wochenschrift*, August 20, 1901, p. 1340; and "Zur Kenntnis des Myeloms und der Sogenannten Kahler'schen Krankeit," *Zeitschrift f. klin. Medicin*, 1902, vol. xlvi., p. 445.)

The patient was a woman, aged thirty-seven years, who was admitted to the Hamburg-Eppendorf Hospital in November, 1900. Her illness commenced with pains in the hips. Afterward she suffered from pains in the loins and sternum, progressive kyphosis, spontaneous fractures of the thigh bones, and increasing debility. She died from pneumonia, one and a half years after the commencement of the illness. During the last weeks of life her urine contained a considerable quantity of Bence-Jones proteid. She likewise had nephritis and hemorrhagic glaucoma. After death an albumose-like body was detected in the blood. Though the authors originally considered that the case was one of genuine osteomalacia, their description of the post-mortem examination gave rise to considerable doubt as to the correctness of their view. Dr. T. R. Bradshaw, in a short communication on the subject (*Münchener med. Wochenschrift*, February 4, 1902, p. 191), enumerated the main objections to the acceptance of their pathological inferences, and the authors afterward republished the case as one of multiple myeloma. It should be noted that in this case the pelvis and long bones were affected as well as the ribs and vertebral column.

No. 25. *Donetti's Case.* (E. Donetti, "Sulla Malattia di Kahler," *Rivista Critica di Clinica Medica*, Florence, 1901, No. 46, p. 789.)

The patient was a man, aged forty-five years, with a painful affection of the bones, especially of the vertebral column and ribs, with anæmia, and with Bence-Jones proteid constantly in his urine. The method of D'Allocco seemed to demonstrate the presence of Bence-Jones proteid in the blood (compare Case No. 15).

No. 26. *The Case of Hijmans van den Bergh.* (A. Grutterink and C. J. de Graaff, "Ueber die Darstellung einer Krystallinischen Harnalbumose," Hoppe-Seyler's *Zeitschrift f. Phys. Chemie*, 1902, vol. xxxiv., p. 393. A. A. Hijmans van den Bergh, "Albumosurie," *Herinnerungs-Bundel Prof. Rosenstein*, 1902.)

The patient, a man-servant, aged thirty-six years, had enjoyed good health until September, 1900, when he was seized with pain on the left side of the chest. Afterward he had pains on the right side of the chest, in the right shoulder, and in the back. He had to give up work, and was admitted into the Rotterdam Hospital in March, 1901. In the hospital great deformity of the thorax occurred from progressive bending of the vertebral column and sternum, and death took place in December, 1901. At the necropsy the bone-marrow of vertebræ, ster-

num, and ribs was found transformed into a sarcoma-like growth. A femur was examined and found similarly affected, but in a lesser degree. The liver was fatty and the kidneys showed slight interstitial changes. Miss Grutterink and Miss de Graaff succeeded in obtaining the Bence-Jones proteid in a crystalline form from the urine of this case. I have to thank Dr. Bradshaw for kindly placing Dr. Hijmans van den Bergh's publication at my disposal.

No. 27. *Conti's Case.* (Pietro Conti, "Albumosuria e Neoplasie Sistematiche delle Ossa," *La Clinica Medica Italiana*, Milan, 1902, pp. 211–247.)

The patient was a lady, aged sixty years, whose first symptoms were costal pains in August, 1899, when she was at the health resort of Mont-Dore on account of bronchial catarrh. She died October 30, 1901, after about tweny-six months of suffering. The symptoms of the skeletal disease were chiefly referable to the bones of the trunk—pains connected with the ribs, sternum, and spinal column; kyphosis; and little tumors on the sternum and right ilium. There was cachexia, and toward the end the muscular weakness was extreme. Bence-Jones proteid was first detected in the urine in November, 1900. For several weeks prior to the death of the patient the urine is said to have contained albumin, but no longer any Bence-Jones proteid; it likewise contained hyaline and a few granular casts. Unfortunately, no post-mortem examination was made. I am indebted to Dr. A. E. Garrod for drawing my attention to Conti's publication.

No. 28. *The Present Case.* (Abstract already given.)[1]

Uncertain Cases and Cases in which the Reactions of the Proteid in the Urine were not Quite Characteristic.

No. 29. Dr. Sidney Martin (discussion on Dr. Bradshaw's paper, *Proceedings of the Royal Medical and Chirurgical Society*, 1898, third series, vol. x., p. 120) referred to the case of a woman under the care of Dr. H. R. Spencer, at University College Hospital, for an ovarian tumor, which was removed. The urine, sometimes milky from precipitation of the proteid, was examined by Dr. Sidney Martin, who states that it contained "the same body or bodies" as those referred to by Dr. Bradshaw in his case. The subsequent history of the case is not given.

No. 30. Dr. R. Hutchison (discussion on the "Proteids in Urine," *Transactions of the Pathological Society*, London, 1900, vol. li., p. 146) referred to a man who died in the London Hospital with multiple tumors of bones (extremities, ribs, and vertebræ). A flocculent precipitate separated out from the urine at 58° C., but did not redissolve on boiling. In this respect and in its behavior to nitric acid the substance present in the urine had not quite the characteristic reactions of Bence-Jones proteid. The case should certainly, however, be men-

[1] Recently Dr. J. M. Anders and Dr. L. N. Boston, Lancet, London, January 10, 1903, have described three cases of Bence-Jones albumosuria, and Dr. Boston has given notes of a fourth, THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, April, 1903. Vignard and Gallavardin, Revue de Chirurgie, Paris, 1903, No. 1, have recorded two doubtful cases. Campbell-Horsfall, Lancet, London, April 25, 1903, has apparently observed *temporary* genuine Bence-Jones albumosuria after a severe injury to the leg. J. A. Milroy's case, Journal of Pathology, Edinburgh, vol vii. p. 95, of multiple myeloma with albumosuria has been unfortunately omitted in making up my list.

tioned here. Dr. Hutchison kindly informs me that the patient was thirty-eight years old, and died within a week of admission. The upper end of one humerus, preserved in the London Hospital Museum, is much enlarged by a very vascular growth. Sections from one of the growths show it to consist chiefly of rather large, rounded, or polygonal cells, with a good deal of protoplasm around a medium-sized nucleus. The protoplasm of many of the cells, Dr. Hutchison tells me, contained granules, possibly of the same nature as those in the cells of the growth in my case.

No. 31. Dr. Lee Dickinson (discussion on the "Proteids in Urine," *Transactions of the Pathological Society*, London, 1900, vol. li., p. 170) mentioned a case of leucocythæmia in the practice of Mr. Edgecombe Venning in which Bence-Jones proteid—or, at all events, a proteid coagulating like Bence-Jones proteid at a relatively low temperature—occurred in the urine, and in which no other disease but leucocythæmia could be found.

Supposed Cases which have been Incorrectly Included in Summaries of Bence-Jones Albumosuria Cases.

No. 32. A case described by Byrom-Bramwell and Nöel-Paton ("On a Crystalline Globulin occurring in Human Urine," *Reports from the Laboratory of the Royal College of Physicians*, Edinburgh, 1892, vol. iv., p. 47) was at one time regarded by Huppert ("Ueber einen Fall von Albumosurie," Hoppe-Seyler's *Zeitschrift f. Phys. Chemie*, vol. xxiii., p. 500) as an instance. But after himself examining the proteid from the urine in question he altered his mind (Ueber den Nöel-Paton'schen Eiweisskörper," *Centralblatt f. die med. Wiss.*, July 9, 1898, p. 481) and regarded the substance as a globulin. The case is remarkable for the spontaneous precipitation of the proteid in crystalline form on allowing the urine to stand for a longer or shorter period, sometimes a day or two, sometimes weeks or months.

No. 33. Simon (THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, June, 1902, p. 954) wrongly cites a case reported by Karl Ewald ("Ein Chirurgisch Interessanter Fall von Myelom," *Wiener klin. Wochenschrift*, 1897, p. 169). The patient was a man, aged sixty-two years. The diagnosis of myeloma was made during life owing to the examination of part of a growth removed from the right clavicle in April, 1894. Death occurred in May of the same year. No necropsy is recorded, and Bence-Jones proteid is not said to have been found in the urine. The case, therefore, cannot be accepted as an example of multiple myeloma with Bence-Jones albumosuria, although Jochmann and Schumm (*Zeitschrift f. klin. Med.*, 1902, vol. xlvi., p. 467), as well as Simon, have referred to it as such.

No. 34. A supposed case of Dr. Vladimir de Holstein (*Semaine Médicale*, 1898, p. 206, and 1899, p. 83) is likewise referred to by Simon, *loc. cit.*, p. 955), who states that the diagnosis of "multiple myelomatosis" was made during life, owing to the state of the urine, and that the diagnosis was confirmed by a subsequent necropsy. However, on looking up Simon's references, I could only find a notice of Bradshaw's case, and a short résumé of the subject by Dr. V. de Holstein, but no new case.

A CONVENIENT MODIFICATION OF TESTS FOR HYDROCHLORIC ACID IN GASTRIC CONTENTS.

By Charles Sumner Fischer, M.D.,
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The modification consists of a simple process of combining three well-known methods in such a manner as to make it possible to estimate both free and combined hydrochloric acid in the smallest possible quantity of gastric contents and within a space of time calculated to make this practical for clinical use. The three methods referred to are those of Töpfer for free hydrochloric acid, of Honigman and v. Noorden for hydrochloric acid deficiency, and the more recent method of Cohnheim and Krieger for the determination of combined hydrochloric acid, which method has not received the clinical attention which it deserves. The desirableness of possessing some more rapid and fairly accurate method for the determination of the combined hydrochloric acid in the clinic must be evident to everyone whose attention has been given to this subject, for the most accurate methods which we possess, namely, those of incineration, require the experience and laboratory surroundings of the trained chemist for their proper operation, and even then their accuracy is a subject for discussion in some cases.

Of these methods, those of Sjoqvist, Leo, Hehner-Seeman, and Martius and Lüttke have survived the test of time, but of them all the last one is probably the only one which can be adapted to practical use by the clinician. Even this method has been subjected to criticism by Rosenheim and requires a certain chemical skill in incinerating to determine the proper moment when the process is completed, a skill which it is not always possible for the physician to acquire.

Of any of the methods proposed for the determination of the combined hydrochloric acid, that with alizarin, as advocated by Töpfer, is probably the most popular. In this method, practical as it is, the end-reaction is often so gradual and indefinite that the results obtained, except for those of great experience, are valueless in most cases. The well-defined play of colors from yellow to red-violet and pure violet, obtained with solutions of acids, disodiumphosphate, and sodium carbonate become ill-defined in fluids containing proteids in any form, as they may be present in the contents of the stomach. A trial with a 1 per cent. neutral solution of egg albumin to which has been added a known quantity of one-tenth normal hydrochloric acid solution (albumin solution 5 c.c., 0.5 HCl solution 2 c.c.) will convince one of this. The quantity of combined hydrochloric acid in such a mixture would correspond to 0.8 c.c. of the normal solution, thus leaving 1.4 c.c. as

free hydrochloric acid. Titrating such a combination with alizarin, the pure violet tint does not appear until the point at which all the free hydrochloric acid has been neutralized has been decidedly exceeded, while the change itself from yellow to violet is most gradual. In gastric contents these inconveniences are markedly increased, due probably to the more complex composition of the same, to the diversity of original color which they possess, or to opacity and cloudiness which result from particles in suspension that cannot be removed by ordinary filtration.

In this connection I would like to call attention to the method elaborated by Cohnheim and Krieger for the determination of the combined hydrochloric acid in gastric contents, for, while not absolutely accurate from a chemical standpoint, it yields results which surpass in exactness any of our possible clinical methods, especially as its shortcomings are known, and any necessary corrections can be made in the results. The method itself is not so well known but that a short description might be in place. Its action depends primarily upon the precipitation of all proteids in solution by phosphotungstic acid. For this purpose Cohnheim and Krieger employed a 4 per cent. solution of calcium-phosphotungstate. Such a solution added in excess to a solution of proteids containing hydrochloric acid in excess will precipitate all the proteids as proteid-phosphotungstate, whereas the hydrochloric acid previously combined with the proteids will enter into solution as neutral calcium chloride. In gastric filtrates which do not contain free hydrochloric acid in excess a certain known quantity of 0.5 normal hydrochloric acid solution should be added and this taken into account in the final calculation. Hence, given the total acidity of the gastric filtrate with phenolphthalein and the total acidity obtained after precipitation with the calcium phosphotungstate solution, we would have a difference which should represent the combined hydrochloric acid.

In a comparison of the results obtained by their method with those obtained by that of Sjoqvist, Cohnheim and Krieger report errors ranging from 5 to 10 degrees. I am convinced that these can be reduced materially by slight modification of method. One source of error lies in the difficulty of securing the whole excess of acid in the filtrate after precipitation of the proteids. For this purpose repeated washing of the residue would be necessary, which in turn would increase the volume of the filtrate to such proportions as to render all color reactions unreliable. Another source of error lies in the fact that in most subacid gastric contents certain proteid products are in solution in excess of the hydrochloric acid present. These products combine with the hydrochloric acid added in excess to secure complete precipitation with the calcium phosphotungstate and help to swell the preexisting combined hydrochloric acid. In order to eliminate the first source of error it has been my custom to avoid washing off the pre-

cipitated proteids by employing only one-half of the known total filtrate and multiplying the result by 2, the customary procedure in the estimation of the total chlorides by the method of Martius and Lüttke. The second source of error, which is confined to gastric contents containing no free hydrochloric acid, can be corrected by first determining the hydrochloric acid deficiency in the gastric contents according to the idea suggested by Honigman and subtracting this deficiency from the quantity of combined hydrochloric acid found. The details of this second modification I shall describe later.

In a long series of comparative analyses of hyperacid, subacid, and anacid solutions, both natural and artificial, by this method and that of Martius and Lüttke, I have found that the error could be reduced to 4 degrees if the suggested modifications were employed, and that the error was usually upon the side of excess rather than deficiency. In many instances the results were alike. As the method of Martius and Lüttke is not faultless, it was difficult in some instances to decide to which to ascribe the difference in the results obtained; but as an error of 4 degrees cannot vitiate the value of either method from a clinical standpoint, it would hardly be necessary to discuss it further.

In order to make it possible to rapidly estimate the combined and free hydrochloric acid in the same 5 c.c. of gastric contents I have combined and modified these three given methods. The solutions necessary for this are the usual 0.1 normal sodium hydrate solution, 0.1 normal hydrochloric acid solution, and the 4 per cent. neutral solution of calcium phosphotungstate. The latter is easily prepared by dissolving 4 grammes of phosphotungstic acid in 100 cubic centimetres of distilled water by boiling and then neutralizing the boiling solution with an excess of calcium carbonate. The boiling should be continued for a few minutes to drive off the carbon dioxide, after which the solution can be filtered into a vessel graduated at 100 c.c., and whatever fluid has escaped by evaporation can be replaced. These three solutions are stable, and can be kept in burettes ready for use.

The operation itself would be the following: 5 c.c. of filtered gastric contents are measured into a small dry beaker and the free hydrochloric acid estimated with dimethylamidoazobenzol. In doing this it would be wise to follow the advice of Hàri not to carry the titration beyond the deep orange tint. The total acidity is next determined by adding phenolphthalein to the mixture and titrating to the limit of reaction. The results obtained being noted, a quantity of 0.1 normal hydrochloric acid solution, corresponding to the quantity of sodium hydrate solution already employed, is added to obtain the original total acidity of the gastric contents. The total number of cubic centimetres of the mixture being known (gastric contents + 0.5 NaOH + 0.5 HCl), the calcium-phosphotungstate solution is added in sufficient

quantity to bring the whole up to 30 c.c. (15 to 20 c.c. usually are necessary). The mixture is thoroughly agitated and allowed to stand three to four minutes. In order to decolorize the same a small quantity of chemically pure animal charcoal is thoroughly stirred into it and the whole filtered through a dry filter into a dry beaker. The animal charcoal not only retains all the color, but also holds back the phenolphthalein, which substance would otherwise render the following titration uncertain. The filtrate is colorless and very rapidly obtained.

Of this filtrate 15 c.c. are measured into a dry beaker and the total acidity determined with the sodium hydrate solution. The result doubled would give the new total acidity of the whole mixture. As phenolphthalein does not give a clear end-reaction in the presence of calcium-phosphotungstate, Cohenheim and Krieger employed for this titration a 1 per cent. alcoholic solution of rosolic acid. Six drops of this added to the 15 c.c. employed will give a yellow clear fluid which turns to red when the acid is neutralized. The titration should be carried out rapidly and to an end-reaction over a white underlying surface. If any uncertainty as to the end-reaction exists it is safer to add a few drops more of the sodium hydrate solution as any error will be largely in favor of finding the total acidity too low. The difference between this last total acidity and that obtained originally with phenolphthalein will represent the quantity of combined hydrochloric acid in the 5 c.c. of gastric contents employed.

The successive steps in the operation, which requires no more than ten minutes, would be the following:

1. Determine the free HCl with dimethylamidoazobenzol.
2. Determine the total acidity with phenolphthalein.
3. Add 0.5 HCl solution in quantity equal to 0.5 NaOH employed.
4. Add sufficient calcium phosphate to make 30 c.c.
5. Allow to stand three to four minutes, add animal charcoal, and filter.
6. Determine the total acidity with rosolic acid.

EXAMPLE.

Employed gastric contents . . .	5 c.c.	
" 0.5 NaOH	4 "	(free HCl = 20, total acid 80).
" 0.5 HCl	4 "	(total acid 80).
" Na_2WSO_4	21 "	
Total number c.c. . . .	30 c.c.	(total acid) 45
Combined HCl = (80–45) . .	35 "	

In order to secure correct results it is essential that hydrochloric acid should be present in considerable excess, hence the method as given can only be employed where such is the case. In gastric contents which are subacid a slight modification is necessary to secure complete precipitation of the proteids. This can be accomplished by the addition

of an excess of 0.5 normal hydrochloric acid, but in so doing it would be necessary to estimate the hydrochloric acid deficiency according to the idea suggested by Honigman, for any proteid substances in solution not combined with hydrochloric acid would immediately combine with the supplied acid and be added to the real quantity of combined hydrochloric acid originally present. For example, if to a known solution of egg albumin only one-half the quantity of hydrochloric acid necessary to combine with all the albumin present is added, and the operation of titration carried on as given above, the final result will represent the quantity of hydrochloric acid which would combine with the whole of the albumin present. It can readily be seen that the same would take place in subacid gastric contents capable of combining with hydrochloric acid without yielding equivalent acid returns. In order to correct the error the hydrochloric acid deficiency should be subtracted from the quantity of combined hydrochloric acid found in the final calculation.

The modified operation would be the following:

1. Determine the hydrochloric acid deficiency with dimethylamidoazobenzol by adding 0.5 normal hydrochloric acid, and note the quantity employed.

2. Determine the total acidity of this mixture with phenolphthalein and 0.5 normal sodium hydrate. This total acidity minus the quantity of hydrochloric acid added would give the original total acidity of the 5 c.c. of gastric contents employed.

3. Add 0.5 normal hydrochloric acid in excess. Sufficient should be added to make the new total acidity at least 40 degrees higher than the original total.

4. Having noted the total number of cubic centimetres in the mixture, add sufficient calcium phosphotungstate solution to make 30 cubic centimetres and proceed as in the original method. The difference between the total acidity obtained by hydrochloric acid in excess and the final total acidity will represent the quantity of hydrochloric acid combined with all the albumin in the 5 c.c. of gastric contents calculated in degrees. This combined hydrochloric acid minus the hydrochloric acid deficiency will represent the quantity of hydrochloric acid originally combined with proteid in the specimen examined.

EXAMPLE.

Employed subacid gastric contents	5 c.c.	
HCl deficiency (dimethylamidoazobenzol)	1 "	= 20
Total acidity (phenolphthalein)	3 "	= 60
Original total acidity	(60–20) "	= 40
Added HCl in excess	3 "	= 60
" calcium phosphotungstate	18 "	
Total acidity (rosolic acid) of	30 "	= 30
Total possible combined HCl	(60–30) "	= 30
Actual combined HCl in gastric contents	(30–20) "	= 10

In order to secure good results it is necessary that all measurements should be made with great accuracy and that all vessels and pipettes should be perfectly dry. The animal charcoal should be chemically pure and should be tested before using. It is especially important not to carry the titration with dimethylamidoazobenzol beyond the deep orange tint, whereas those with phenolphthalein and rosolic acid should be carried to the full limit. Operating in this way the error can be reduced to 4 degrees, usually as excess of combined hydrochloric acid, and the necessary allowance can be made. As 4 degrees represent a quantity of hydrochloric acid equal to 0.146 per 1000, it is evident that the method is sufficiently exact for most purposes, and requires but little more time than the determination of the free hydrochloric acid and total acidity as usually practised in offices and clinics.

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TWO CASES OF RELATIVE AORTIC INSUFFIENCY.

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and

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Relative aortic insufficiency, by which is meant an incompetency of the semilunar valves without an organic lesion of the valves, is so rare that no excuse is needed for recording two such cases which occurred in the Medical Department of the Royal Victoria Hospital.

Edwards,[1] in 1895, stated that aortic is the rarest of all relative insufficiencies, and that the combined literature of England, France, and Germany did not record more than about twenty-five cases, while some of these were reported twice, though dearth of detail precluded their detection. In the modern text-books there can be found but scant if any reference to the condition.

Case I.—J. H., aged fifty-nine years, paper cutter, was admitted December 15, 1902. About three weeks before admission he com-

[1] The American Journal of the Medical Sciences, vol. cx. p. 438.

plained of cough, dyspnœa on exertion, palpitation, and œdema of the lower extremities, and, according to his statement, an attack of sudden dizziness was followed by paralysis and inability to speak or move his extremities for some hours. Alcoholism and syphilis were denied.

The examination of the patient on admission gave evidence of disease of the circulatory system—dyspnœa, cyanosis, and œdema being present. The pulse was irregular in volume and rhythm and of high tension. There was general arteriosclerosis. The heart was enlarged to the left and right, with a diffuse impulse in the fourth, fifth, and sixth intercostal spaces, one inch outside the left nipple line. At the apex the first and second sounds were not clear. Over the aortic area a systolic murmur was audible and transmitted up the vessels of the neck, while at the same area a diastolic murmur, with transmission down the left border of the sternum, was also heard.

Seven days after admission to the hospital the patient became much weaker. There was orthopnœa, profuse bloody expectoration, and cardiac irregularity. The diastolic murmur was still present over the aortic region. The lungs gave evidence of œdema, and after several hours of distress, marked by the above symptoms, the patient died.

A diagnosis of myocarditis, aortic endocarditis, with regurgitation, mitral endocarditis, with regurgitation, and acute pulmonary œdema was made.

An autopsy revealed the following conditions with respect to the circulatory system: Hypertrophy and dilatation of the heart; arteriosclerosis; relative insufficiency of the mitral valve and aortic insufficiency, apparently secondary to atheroma of the arch.

The aortic valve cusps were unaffected by sclerosis, while the arch and whole of the aorta presented signs of arteriosclerosis in extensive calcification and ulceration. The first part of the arch was markedly dilated, and the insufficiency of the aortic valves was manifestly due to the dilatation of the aortic ring.

For permission to report the above case we are indebted to Dr. James Stewart, and for a report upon the post-mortem findings we are under deep obligation to Dr. Adami.

Case II.—G. R., aged fifty-five years, storeman, was admitted on July 17, 1902, complaining of "kidney trouble," palpitation, and weakness, with faintness on exertion.

He was a large, well-nourished man of fair complexion, the skin of pronounced lemon-color, and the mucous membranes very pale. The patient admitted that many years ago he had used alcohol to excess. At the age of eighteen he had pleurisy on the left side; at the age of thirty, acute articular rheumatism; when fifty-four he had typhoid fever. Further, for the past five years he has been the subject of frequent micturition and burning sensation in the hypogastrium if micturition were delayed. For several years sudden stoppage of the stream of urine was often noted by the patient.

An examination of the various systems led to a diagnosis of arteriosclerosis, emphysema, and cystitis with anæmia, conforming to the type of secondary anæmia rather than to that of pernicious anæmia, for when the anæmia was most profound the hæmoglobin value was 13 per cent., with a red cell count of 1,140,000. Careful microscopic exam-

ination of stained specimens of blood failed to discover the more characteristic varieties of blood cells found in pernicious anæmia.

In examining the circulatory system one found that dyspnœa and palpitation were very marked on exertion. There was slight cardiac enlargement. The heart sounds at the apex were weak, the first sound being very distant. A soft systolic murmur was heard at the apex, but was not transmitted. At the base both sounds were distant, and over the pulmonary area a loud systolic murmur could be heard.

While under observation in the hospital the patient failed to improve with rest, nourishing diet, and hæmatemics, such as Fowler's solution and sodium cacodylate. He gradually grew weaker.

Seven days before death, when weakness was profound, a soft diastolic murmur was first observed, with its maximum intensity at the fourth left intercostal space, but plainly heard at the second right costal cartilage and not heard at the apex. On the following day this murmur was yet more marked, but owing to the patient leaving the hospital further opportunities of examination were not possible. Without any other symptoms the patient's strength failed, and he died within a few days.

Through the kindness of Dr. Anderson an autopsy was obtained and the following conditions were found: Chronic cystitis, with a polypoid tumor about the size of a robin's egg springing from the trigone of the bladder. This, no doubt, accounted for the sudden stoppage of the stream as previously noted. The ureters were dilated, and there was marked double pyonephrosis.

The heart was dilated with some hypertrophy of the left ventricle. Over the right ventricle anteriorly was a small milk patch the size of a five-cent piece. Early and *slight* atheroma of the aorta was noticed. There was no endocarditis. The aortic cusps showed no thickening, nor was there any marked evidence of dilatation of the first portion of the aorta.

Summarizing these cases, with respect to the condition about the aortic valves, we had to deal clinically with a diastolic murmur having all the characteristics of that due to local organic change—a murmur which clinically appeared to arise from no lesion other than that of the semilunar valves—while anatomically we had to deal with cases in which the semilunar valves were perfectly normal in structure. Thus it would appear that we have two genuine cases of relative aortic insufficiency.

Studied in detail these cases show two ways by which this condition may be brought about.

In the first instance, as a result of advanced atheromatous degeneration of the aorta, dilatation of the first portion occurred, rendering the valves, though perfectly normal, relatively incompetent. But while in this case the semilunar valves were found to be insufficient, in the other instance the post-mortem examination showed no leakage whatever at the aortic ring; so that the local changes in the aorta were not sufficient in themselves to account for the clinical phenomena.

In this case, apart from insignificant atheromatous changes in the

first part of the aorta, there were no local organic lesions, but only a pale, flabby condition of the myocardium, such as one would expect to find in association with the anæmia from which the patient had suffered. It is true there was also present a small milk patch on the anterior surface of the heart, but this was apparently of long standing, while it will be remembered the diastolic murmur was discovered but a few days before death.

It is obvious, therefore, an explanation must be sought outside of this, and apart from the causes responsible for the condition in the first instance. The explanation here, doubtless, is to be found in an asthenic condition of the heart muscle, especially of that portion falling under MacCallum's[1] fourth division, which "encircles the left auriculoventricular ring and the aorta."

It may be of interest to recall in this connection that Osler points out, on Beneke's authority, that "the aortic orifice, which at birth is 20 mm., increases gradually with the growth of the heart, until at one-and-twenty it is about 60 mm. At this it remains until the age of forty, beyond which there is a gradual increase in the size up to the age of eighty, when it may reach 68 to 70 mm." Thus there is, at the very period of life in which sclerosis of the valve is most common, a physiological tendency toward the production of relative insufficiency.

In reviewing these two cases it was not our intention to go further into the various causes of relative aortic insufficiency. This has been fully discussed in the article already quoted. Nor was it intended to discuss the causes of diastolic murmurs other than those originating in or at the aortic orifice. This point is fully dealt with in the same article. Our purpose will have been served if these two cases may find a place beside those already presented.

THE COMMUNITY AND TUBERCULOSIS.[2]

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of New York.

Throughout the United States a fair appreciation is now awakened to our duties toward consumptives. This is shown by broad and enlightened work of health boards, by stimulating help from many physicians who have strongly urged their needs, and by contributions of money and effort by laymen and women to build sanatoria.

The sanatoria are of two kinds essentially. First, those where the relatively well-to-do may secure the best care and treatment now recog-

[1] Wood's Reference Handbook of the Medical Sciences, vol. iv. pp. 578–79.

[2] Read before the Climatological Association (to open discussion), Washington, May 14, 1908.

nized, for which, as a rule, a suitable pecuniary return is made. Second, those where the poor, homeless, houseless, friendless patient goes ultimately, to end his days with greater comfort to himself and with less risk to relatives, friends, and people generally. In the latter instance, and as much as may be, provision is also made to receive incipient and slightly advanced cases, which are the more acceptable ones, as they are by far the more curable. In the private sanatoria provision is especially made for the latter sort of cases, and apparently for much the same reason. Within very restricted limits accommodation is also provided for the care of those who are absolutely poor. What has been done by us hitherto for the dwellers of our tenement houses is very little compared with what has been accomplished abroad—in Germany, France, England—and as nothing, in view of the great and urgent necessities of many thousands of diseased. Happily, some munificent donors have already arisen, and in the Phipps endowment notably we appreciate with gratitude the spirit and doing of generous and instructed private philanthropy. In appeals to State Legislatures and municipalities by boards of health, by physicians, and by citizens for sanatoria to be put under judicious public control, and solely for the benefit of the very poor, we recognize the advent of a better era for very many tuberculous patients hitherto relatively neglected or ignored. Adequate provision doubtless will be made in the near future in the same or different institutions for the best care of such patients, according to the stage of the disease.

To me, the idea of sanatoria for the very poor brings joy and gladness, because I see in it prospect of cure for many; prospect of consolation for many more. To be sure, it will, perhaps, be a long while before we may legitimately hope, even with large and numerous public sanatoria buildings, to meet all requirements of the needy consumptives. But with their existence we shall have accomplished a great and noble, though necessarily limited work, and we shall be able effectively to educate a large and increasing number of sufferers; so that when they return to their homes, stationary, improved, or cured, they will know better how to care for themselves and how to protect others.

As to private sanatoria, especially for the favored ones, and with one or two notable exceptions, I frankly confess I do not feel that the return of health and strength to relatively few patients justifies wholly the outlay of money, labor, notoriety, in behalf of these institutions. I may be misinformed or in error; but in many ways I believe such patients, with their own physicians and with large, sunny, well-ventilated rooms, appropriate surroundings, good food, care, and discipline, in a well-selected location in the country, would enjoy advantages not fairly counterbalanced by excessive *doing* and *overminute* regulations (often calculated to do more harm than good) of private sanatoria.

What shall I say of home treatment? To the vast number in our midst at present it is the only available treatment. Even if we could persuade very many among the poor to leave their homes and go elsewhere, there is no institution yet open to receive them. In the city of New York there is now accommodation approximately for 1000 of her consumptive poor; there is requirement for 10,000 to 20,000 more! Fortunately, the day of specifics is passing for all honest and intelligent practitioners. It is useless for enthusiastic or overzealous men to laud and magnify (after very moderate or wholly insufficient experience and cases) this or that drug, this or that preparation or means of cure. Hygiene in its broadest, best sense is, after all, our main reliance; and statistics based upon other means, no matter how favorably interpreted by their warm advocates, meet with little cordial response from those who have learned by long experience to become properly skeptical. Most of us now look forward to the bright day when, perhaps, a really efficacious antituberculous serum shall be discovered—not a mere destroyer of bacteria, and perhaps with this destruction great or irremedial injury to the blood or other fluid of the economy—but rather to a serum which shall quickly neutralize or destroy those hitherto unknown toxins, of which the bacteria are but the formed and visible evidence of their presence and action. Better still. May we not even legitimately hope that a serum will be discovered which shall produce, when inoculated, almost absolute immunity from the development of tuberculosis? Surely, the admirable researches of Dr. E. L. Trudeau, at his Saranac Sanatorium, point in the not distant future, perhaps, to the practical realization of this great hope for the race.[1]

What we want, what we insist upon for our poor, is better ventilation, more and better air, more sunlight, improved plumbing, greater cleanliness in their homes. To these essentials we add good food, properly prepared and served; a pure and plentiful supply of milk and water. Much of all this means of necessity, therefore, improved tenement houses, and under no possible pretext to allow any unjust, iniquitous laws to be passed which would interfere with our ideals or oblige us to take a step backward in our march of progress and greater civilization.

What shall we gain by following out such a plan? First of all, we shall secure a greater resistance on the part of a susceptible or already diseased individual to attack or further inroads of disease; and in this way we shall be doing much toward the ultimate inhibition of tuberculosis. Such an outline as that given above may be helped by judicious medical treatment, and this may be effected in the homes and in the dispensaries.

[1] See paper by Flexner in Philadelphia Medical Journal, February 14, 1903.

My statement has been proven already, I am glad to say (in a limited number of cases up to the present time), by Dr. John F. Russell, of New York City, at the Post-graduate School and Hospital. Dr. Russell supports his patients by the administration of various fats in assimilable form, and by due attention to the condition of the primæ viæ, while they are submitted at the same time to frequently repeated feeding of appropriate kind. In this way he adds still further to the capital *of resistance in the individual patient*, which should be our primary object to develop, foster, and maintain. Many of Dr. Russell's patients have recovered; many more, as the years roll on and the number of his patients increases, will recover. Dr. Russell's ideas are based somewhat, doubtless, upon the facts fully made known to us twenty-five years ago by the late Professor Austin Flint—*i. e.*, that pulmonary tuberculosis was a self-limited disease (taking all cases), in the proportion of 1 to 15, and even in the midst of unsanitary surroundings, which up to the present time, unfortunately, are so usually the lot of the poor. It is only rational, therefore, to believe that with improved surroundings, and better care and treatment, a far larger proportion would recover.

In our efforts, however, to improve or abolish the nests of disease we must never lose sight of the broadest humanity for the sufferer. Do not make him a social outcast! He is already sufficiently stricken. Do not add to his misery! Many patients who are unquestionably phthisical may still do light and healthful work and continue to improve physically, and, indeed, ultimately recover. Is not this fact of immense importance? And not merely because it keeps hope and courage alive, but also because, from a material and purely economical standpoint, it saves much money to the State, which otherwise is morally bound to be the caretaker and supporter of the doubly afflicted poor.

But it is often proclaimed that such people—the tuberculous—are a constant imminent danger to others—to the well ones. This is not true if the patients affected with pulmonary tuberculosis be properly instructed and disciplined in their homes and places of employment. It cannot be too strongly insisted upon and too widely circulated that tuberculosis *is not contagious* in the ordinary sense of this word, and in the same way the eruptive fevers, for example, are contagious, through a third person or through the air of the patient's room. One may be as safe in the room of a phthisical patient as anywhere else, provided, of course, every rational and important precaution and observance be upheld to prevent infection. The disease is infectious and communicable—*that* we know; but, once more, this statement does not mean that it is *contagious*.

Viewed differently, what do we see? The crudest expressions of dire ignorance on the one hand, and extended inhumanity on the other.

People who have a foolish, unreasonable dread become immediately unreasoning, unkind, inhumane, un-Christian. In certain of our States and Territories they have wished to enact laws to prevent consumptives from entering their borders. The National Government has even been requested to prevent consumptives from coming to the United States from foreign countries, and only so far back as 1901 the Treasury Department of the United States decided to classify pulmonary tuberculosis with dangerous contagious diseases and to exclude all consumptive immigrants from entering the United States. Thanks to the intelligence, broad-mindedness, and, above all, the humanity of the medical profession, this order was strongly opposed. Thus far, unfortunately, their laudable efforts have remained without effect upon the powers that be.

On February 20, 1903, I received a letter from the Commissioner of United States Immigration Service stating that "the statute is enforced in accordance with the opinion" expressed by the Surgeon-General of the Marine-Hospital Service—*i. e.*, that "tubercle of the lung is a dangerous contagious disease."

In the Adirondacks, where formerly consumptives encountered only affection, love, care, attention, sympathy, in many instances they are now almost outlawed at times from getting proper shelter and food, and simply because the "poor blind ones" imagine they will contract a terrible disease by reason of contact. This leads me to say what I believe to be very important—that after all hygienic rules are obeyed and all precautionary rules laid down and insisted upon, to protect the healthy and also to prevent reinfection of the diseased it is essential among the poor to provide proper disinfectants, spit-cups, and paper napkins for sputa. In the outline of home treatment in the new Phipps Sanatorium this rule of action on the part of physicians or care-takers of the poor is duly and forcibly emphasized.

Finally, the broad statement still remains true, that without a susceptible soil no tuberculosis will develop in the vast majority of cases. Witness a little experience of my own. I served thirteen years in the Out-door Department of the New York Hospital, where I had the class either of heart and lungs or throat and nose. During that time I had three or more assistants at different times, and several students. I or my assistants passed at least two hours three times a week in the room where the patients were examined, treated, and prescribed for. In no instance that I recall was anyone among my assistants or students (of course, including myself) known to have contracted tuberculosis, and certainly in no instance was our attention directed to the room of the New York Hospital as being the source and origin of the disease.

So far as I know, at no time in the thirteen years referred to above was the room where I examined and worked over my cases ever thor-

oughly disinfected in accord with the notions that we now possess of the meaning of the term. Of course, patients coughed, expectorated, and frequently tuberculous sputa reached the floor, dried up, and were wafted freely later to the respiratory tract of us all.

On the other hand, with a susceptible soil, the *development of the disease is very largely preventable*, always supposing that we insist upon the observance of rules, with this object in view, which we know and believe to be necessary. Shall we rid ourselves entirely of pulmonary phthisis? Perhaps not very soon, in view of all the numerous bad conditions of modern life, especially in our great centres of population and among the very poor, who are obliged to start in life, being the possessors of that hereditary susceptibility to disease which proceeds from one or more generations, probably, of corroding care, unrelaxed labor, lack of proper lodgings, food, air, and sunlight. But with improvement, amelioration, a far better state of affairs is sure to come.

Suppose this to be an "ignis fatuus"—as some may call it—we may even then at least hope that "probably, in future years, tuberculosis may be a comparatively rare and negligible ailment; but if this is to be, it will be through rational and humane methods, and not reckless exaggeration and stimulation of insane public fears, with their natural result of brutal inhumanity to the unfortunate."[1]

To show to what lengths this inhumane spirit even in our profession may go I have only to cite the writer of an article, lately published in one of the leading New York medical weeklies,[2] who appears to charge many consumptives "with a deliberate criminal tendency to spread their disorder."[3]

To show the other side of the picture and the one we would dwell upon, believe, and know to be true, I will cite from an article in the current issue of *Charities* (February, 1903):

"There is at present," the article states, "no other disease which is receiving so much attention at the hands of social and philanthropic workers. One reason may be that its cause is so well defined and the methods whereby its spread can be prevented are so simple that they may be easily grasped by the public. There is reason to believe that within a comparatively short time the United States will have as complete an organization for the prevention of tuberculosis as any to be found in Europe."

To my mind, tent life is the perfect life for consumptives or those threatened with the dread disease, for in this way, and in this way alone, may they keep their lungs filled all the time with the best air possible; and by adoption of this method of treatment we have a guar-

[1] Journal of the American Medical Association, November 15, 1902, p. 1259.
[2] New York Medical Journal, February 8, 1902, p. 238.
[3] Journal of the American Medical Association, loc. cit.

anty of cure in certain cases where it appears to be almost essential, if this most desired result is to be attained. For this reason I believe, independently of the questions of less expense and larger numbers provided for, the New York Board of Health has wisely determined to adopt the method of tent treatment of the consumptive poor in the incipient or primary stage of disease, whose cases it may be able later properly to care for.

No doubt, also, the amount of exercise judiciously proportioned in individual cases, which to many a camp life almost necessitates, is additionally a reason why tent life is an ideal life for these sufferers. Keeping the tent "shipshape," or keeping the camp cleanly, orderly, attractive, is surely useful and beneficial work for them. Their minds are agreeably occupied, and their bodies freshened and strengthened in performing the tasks, with this object in view, duly assigned to them.

Except in the febrile exacerbations of the disease, or in cases of extreme weakness, there are few instances in which tuberculous patients will not obtain distinct advantages from this mode of life over any other, provided, of course, the tent or camp life is followed in a desirable locality and with ambient surroundings in every way hygienic.

Latterly, Dr. A. Mansfield Holmes[1] called particular "attention to the essentials of an ideal tent cottage, and gave rules for governing tent life." . . . . "A model of the tent cottage adopted by the Rocky Mountain Industrial Sanatorium was exhibited" by him before the Mississippi Valley Medical Association, "showing improved methods of construction and ventilation."

AN EPIDEMIC OF TYPHOID FEVER DUE TO IMPURE ICE.

By R. H. Hutchings, M.D.,
AND
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The St. Lawrence State Hospital is situated on the St. Lawrence River, three miles below the city of Ogdensburg. At this point the river is a mile and a quarter wide, has an average depth of from twelve to twenty feet, and the current is three miles per hour. This was the source of the water for the hospital from its organization in December, 1890, until December, 1900, the intake pipe extending two hundred feet from the shore into swift water, and to a depth of sixteen feet, on a rocky bottom. The water supply was considered ideal by the founders of the institution, who pointed to the great volume of the river, the

[1] New York Medical Journal, February 21, 1903, p. 346.

comparatively rapid current, and the rocky formation of the river bed, and argued that the danger of contamination from the Ogdensburg sewers was reduced to an infinitesimal quantity; yet, within two months from the occupation of the first building, typhoid fever made its appearance and was practically endemic for ten years, increasing steadily with the growth of the population from two cases the first year to forty in 1900. In September, 1895, at the request of the superintendent, the State Board of Health made an investigation of the prevalence of typhoid fever in the hospital, and the report by Dr. F. C. Curtis embodied the following conclusion: "I would report that it is my opinion that this water supply (the St. Lawrence River) is the source of the endemic. 1. Because the other common sources for the development and spread of typhoid fever may all be excluded. 2. Because the characteristics of the endemic are those of such a constantly acting cause. 3. Because in the vast majority of cases the epidemic or endemic prevalence of typhoid fever is due to a contaminated water supply. In the present case we have a navigated and sewage-bearing stream used as the source of supply." Previous to this investigation the water had been tested chemically and bacteriologically with negative results. All observers agreed in the correctness of this conclusion, and it is not likely that ice at that time contributed in any appreciable degree as a causative factor. There are several reasons for this belief, one of which will suffice here, and that is in the earlier years each ward had its own drinking fountain, where the water was cooled in an ice chamber in the basement, and ice was not put into water or milk.

Boiled water was then provided for drinking and every effort made to prevent employés and patients using water from the faucets for that purpose. By this means the disease was kept in check to some extent; but employés would become careless after a time when there was no sickness to remind them of it, and many of the patients, from delusions of poisoning or for other reasons, refused the boiled water and drank from the faucets in the lavatories.

Various measures for purification were investigated, and it was finally decided to abandon the St. Lawrence River and obtain water from the Oswegatchie River, a small Adirondack stream, from which the city of Ogdensburg is supplied, by extending our mains to connect with those of the city. This was done in December, 1900, and with the exception of two cases which occurred about the time the change was made, and probably contracted previously, there were no cases of typhoid that were not clearly contracted elsewhere until October, 1902. During this period of twenty-one months water was not boiled and ice was freely used as an article of food by both patients and employés.

On October 2d three attendants in the same building were reported ill with fever, headache, and malaise; on the 3d, two others with like

symptoms; on the 4th another case occurred; on the following day two others, making eight within four days. The symptoms presented by these early cases were principally of a nervous type; there was intense aching in the back and limbs, insomnia, headache, restlessness, and dry cough. The temperature of several of the patients reached 103° to 104° F. the first day.

The disease was considered epidemic influenza until toward the end of the first week, when the rash and other symptoms of typhoid made the diagnosis no longer doubtful. Of the eight persons first attacked, seven were employés who ate in the same dining-room, and the other was a woman patient who had been working there. The local conditions about this dining-room and kitchen were thoroughly inspected, with negative result; the milk is obtained from our own herd and could not have been infected. All the containers were sterilized twice daily, no water was used except from the faucets, and there had been no fever among the men working in the barns. The possibility of infection from vegetables and oysters was eliminated after investigation. Inquiry of the health department of the city showed that typhoid was not prevalent there among users of the Oswegatchie water. There remained only the ice to be considered, and one point of interest in this connection is that about six days before the first cases developed a new ice-house had been opened from which none had been previously withdrawn this year. The ice had been taken from the St. Lawrence River and had been stored since February, a period of more than seven months, and though gathered from the same spot as that in house No. 1, it was at a different time. Dr. John W. Benton, health officer of the city of Ogdensburg, informs us that during the months of January and February, 1902, when this ice was forming, there were three or four cases of typhoid fever in the city among users of well-water. None of them was verified by autopsy, but he thought there could be no question as to the nature of the disease. The temperature prevailing during the week the ice was being stored was, according to the records of the Weather Bureau observer at this point—maximum, 14° F.; minimum, —1° F. The ice was cut from the same spot during the past twelve years but one, and was never before suspected of conveying disease.

A specimen of drinking water and one of melted ice was sent at once to the Bender Laboratory in Albany, N. Y., for bacteriological examination. The ice selected was clear and free from extraneous matter. The report of the director, Dr. George Blumer, is in part as follows: "Specimen No. 1, drinking water (Oswegatchie). Number of bacteria per cubic centimetre on agar plates, 11,000; on gelatin plates, 3600. The fermentation test showed no gas in eight tubes, each treated with one cubic centimetre of water. Specimen No. 2, melted ice (St. Law-

rence). Number of bacteria per cubic centimetre on agar plates, 30,400; on gelatin plates, 50,400. Out of eight fermentation tubes inoculated three showed definite evidence of organic contamination in the form of the colon bacillus." This report from so excellent an authority satisfied us that the contagion of typhoid was in all probability in the ice from ice-house No. 2, and we began an investigation in the laboratory of the St. Lawrence State Hospital to determine whether or not the bacillus typhosus could be isolated from it.

We examined the stock of ice, and found that some of the cakes contained foreign substance in the form of black or dark brown, granular matter, solidly frozen in the ice. These cakes were broken and the portions containing the foreign matter removed. The cakes were about sixteen inches in thickness, and the portions selected were, as near as possible, from the centre. We were careful to choose no snow ice. These fragments were melted in a clean vessel at room temperature, after which a considerable black sediment was deposited in the vessel. Examined under the microscope with low power it appeared as black, homogeneous masses; with the $\frac{1}{16}$ objective the field was crowded with organisms, many of which were motile. Cultures made from the sediment and the water, in bouillon, showed a rapid growth, with fecal odor in all the tubes. Series of plates were then prepared from these cultures by the usual method, and well-defined colonies were isolated in the end plates.

Five colonies were selected which presented characteristics resembling the typhoid bacillus, and fresh cultures were made from them. Of these cultures three proved to be the colon bacillus, one was a bacillus not identified, while the fifth was a pure culture of the typhoid bacillus, as shown by the following tests: On nutrient agar it grows readily; in broth no pellicle is formed; in lactose media no fermentation occurs; on potato the growth is invisible; in litmus milk the reaction is faintly alkaline and no coagulation occurs; with the serum of typhoid fever patients characteristic clumping is produced; it is an actively motile bacillus having the appearance of the bacillus typhosus. The disease was identified clinically by autopsies in three of the cases, in all of which the intestinal and abdominal lesions were demonstrated.

In "An Investigation of the Boston Ice Supply,"[1] in 1901, conducted by the board of health, the work of Sedgwick, Winslow, and Park is reviewed and the following statement made: "These considerations show that ice more than three weeks old is sanitarily as safe as a well-filtered water supply. Cases of typhoid fever, due to ice, might naturally be expected to occur, if at all, at the time immediately following the cutting of infected ice, and as this is done in

[1] Boston Medical and Surgical Journal, vol. cxlv. p. 557.

January usually, after March at latest the ice could hardly be held responsible for any trouble, even were it known to be infected." It should be borne in mind, however, that the work of the authorities referred to and upon which this conclusion is based was done under artificial conditions, and in its natural environment the bacillus now appears to have a greater resistance, not heretofore recognized. In our review of the literature we have been unable to find any similar cases reported, and in the article quoted the statement is made that only a single authentic case is on record.

With the discontinuance of the use of the infected ice the epidemic gradually subsided. There were in all thirty-nine cases, the last of which developed in November. Our water is not boiled, and the conditions, with the exception of the ice, are the same as at the beginning of the epidemic.

THE TRANSMISSION OF BOVINE TUBERCULOSIS BY MILK, WITH A TABULATION OF EIGHTY-SIX CASES.

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The agency of milk in the causation of scrofula was referred to by Carmichael[1] in his *Essay on the Nature of Scrofula, with Evidence of its Origin from Disorders of the Digestive Organs*, London, 1810, in which he refers to *acescent* (sour) diet, especially of cow's milk, as a frequent cause. On page 50 he says: "Infants at the time of weaning are from the change of diet particularly subject to bowel complaints, too often followed by disease of the mesenteric and lymphatic glands."

He quotes Richard Wiseman (1626–1686), White (1787), and Lieutand to show that whenever the outward glands appear swollen, one may safely conclude the mesenteric to be so, too, they being usually the first part that is attacked by this malady. Indeed, Carmichael, while he never saw a subject in the dissecting-room with strumous glands externally without a similar state of those of the mesentery, claims that he has very frequently seen the latter unaccompanied by any affection of the external glands, which to his mind indicates that in scrofula the mesenteric glands are the first involved.

While Carmichael did not attempt to prove the transmission of scrofula in the milk of diseased cows, he points with emphasis to the use of sour milk, and on page 101, remarks:

"With respect to the disease in question, we know that swine are so subject to one very similar that scrofula has in consequence derived its name

from those animals, and certainly their extraordinary fondness for *acescent* food corroborates in some degree the foregoing opinion. Swine, it is well known, fatten upon buttermilk and upon the sour liquid formed in starch manufacture during the steeping of wheat."

These are suggestive statements, especially as, in the opinion of veterinarians of the present day, tuberculosis is increasing among swine, largely as a result of consuming tuberculous milk. According to Salmon,[2] during the year 1900, of 23,336,884 hogs slaughtered under Federal inspection, 5444 were sufficiently affected to cause condemnation of some part of the carcass.

Casper,[3] in his *Characteristics of French Medicine*, etc., published in 1822, on page 124, writes:

"Scrofula is not more rare in Paris than elsewhere, and baffles also here the efforts of physicians. La Billardière declares that the majority of milch cows in Paris perish from nodular consumption, and that their milk contains seven times more of lime phosphate than common. It is possible that there is a connection between this phenomenon and the many tuberculous diseases among the children in Paris. We cannot pursue here this investigation, where the mere fact suffices that in Paris, especially in the hospitals for foundlings and for children, like the St. Louis and others, the sequela and effects of scrofula can be seen in astonishing numbers."

La Billardière's[4] statement that the milk of tuberculous cows contains seven times more of lime phosphate than common is of special interest in view of Dr. de Schweinitz's[5] recent biochemical researches upon bovine, swine, avian, virulent human, and attenuated human tubercle bacilli, in which he calls attention to the large amount of phosphoric acid obtained from the germs, and also points out that the conclusions which might be drawn from these analyses indicate a closer resemblance in the composition of the germs between the moderately virulent human and the very attenuated human bacilli.

Klencke,[6] in 1846, was the first to write on the infectiousness and transmission of scrofula by cow's milk. In his book of ninety pages he gives the clinical histories of sixteen children who had been fed with the milk of scrofulous and tuberculous cows, and they all point to tuberculosis of either the intestines, glands, skin, or bone. He also describes the condition of the seven suspected cows, and verified the diagnosis of a "scrofulous-tuberculous condition" in four post-mortem examinations of the cows, which demonstrated to his satisfaction "that the same scrofulous lesions may be developed in the cow as are found in the human subject and that human scrofula and the disease found in the mammalia are identical." The details of these cases were republished by the writer in 1895.

Klencke's contribution to the early literature of this subject may be regarded of special value, as he was one of the first to demonstrate the

infectious nature of tuberculosis by injecting, in 1843,[7] human tuberculous matter into the cervical veins of rabbits, causing pulmonary and hepatic tuberculosis in these animals. In passing it should be stated that inoculation experiments had been made by Kortum (1789), Hébréad (1802), Salmade (1805), Goodlad and Deygallières (1829), Lepelletier (1830), and Laennec and Erdt (1834[8]). These experiments naturally paved the way for Villemin's[9] brilliant and positive demonstrations in 1865.

Since that time numerous instances of artificial tuberculosis have been reported, the experiments having been performed on heifers, calves, goats, swine, guinea-pigs, rabbits, and dogs; so, for instance, under the heading of *subcutaneous inoculation methods* may be mentioned the work of Soujou and Court Paul,[10] Gerlach,[11] Günther and Harms,[12] Rivolto and Perroncito,[13] Bagge,[14] Zürn,[15] Bollinger,[16] Biffi and Verga,[17] Bouley,[18] Aufrecht,[19] Toussaint,[20] and others. *Intraperitoneal inoculations* were reported by Klebs,[21] 1870, Bollinger,[16] 1873, and Kitt,[22] 1878. *Intravascular inoculations* were reported by Semmer, Thal, and Nesterow.[23] *Intraocular inoculations* were reported by Cohnheim and Salomonson,[24] Hänsell and Deutschmann,[25] and Baumgarten;[26] and finally the *inhalation experiments* by Tappeiner,[27] Lippl, Reinstadler and Bertheau[28] may be cited.

It is true that opposing views were held and quite a number of negative experiments have been reported, but on the whole the results were sufficiently numerous and uniform to demonstrate the identity of scrofula and tuberculosis and the intercommunicability of tuberculosis in animals and man. This evidence was materially strengthened by the feeding experiments of Gerlach,[29] Chauveau,[30] Bollinger,[31] Klebs,[21] Zürn,[15] Roloff,[32] Brell,[33] Metzguer,[34] Langeron,[35] Blumenberg,[36] Orth,[37] Lange,[38] Peuch,[39] Peuch and Toussaint,[40] Aufrecht,[41] and others.

Bollinger[42] in the discussion of artificial tuberculosis before the German Naturalists and Physicians in 1879 (which was participated in by v. Recklinghausen, Arnold, Birch-Hirschfeld and Fränkel) had no hesitation in declaring that milk from tuberculous cows is largely responsible for the production of scrofula, which affects about 70 per cent. of the children in Munich.

E. Klebs,[43] from his experiments in 1871–1873, concluded that milk from tuberculous cows produces tuberculosis in different animals; that the disease commonly begins with an intestinal catarrh, followed by tuberculous affections of the mesenteric glands, involvement of the liver and spleen, and finally by extensive miliary tuberculosis of the pulmon-

ary organs, and Virchow,[44] in 1880, suggested that bovine tuberculosis may be conveyed in the milk, especially if the udder be the seat of the lesion. In the meantime a number of cases of transmission of bovine tuberculosis in milk had been reported by Schoengen,[45] Leonhardt,[46] Bollinger and Stang,[47] Demme,[48] Epstein,[49] Uffelmann,[50] Johne,[8] and Law.[51] Creighton,[52] in 1881, published a book on bovine tuberculosis in which he presents a minute study of a number of cases of tuberculosis in man which had the characteristics of an infection and at the same time a suggestive likeness in the morbid products to the bovine form of tuberculosis (*perlsucht*). Indeed, the evidence on this subject appeared sufficiently strong to justify Koch,[53] in 1882, in declaring:

"It is not the peculiar structure of the tubercle, nor its lack of blood-vessels, nor the presence of giant cells, that will give the solution, but rather the proof of the tuberculosis bacillus, whether it be in the tissue by means of staining reaction or whether it be by means of culture upon coagulated blood serum. This criterion being adopted as a foundation principle, according to my investigations miliary tuberculosis, caseous pneumonia, caseous bronchitis, intestinal and glandular tuberculosis, bovine tuberculosis, spontaneous and inoculation tuberculosis of animals must be declared as identical. . . . Bovine tuberculosis is identical with human tuberculosis, and, therefore, a disease transmissible to man. It should be treated just like other infectious diseases transmissible from animals to human beings. However great or small may be the danger which results from the consumption of meat or milk affected with bovine tuberculosis, it is present, and must be, therefore, avoided.

Soon after Koch's discovery of the tubercle bacillus diligent search was made for the presence of this organism in the milk of diseased cows, and it was demonstrated by Csokor,[54] Bang,[55] Johne,[56] Bollinger,[57] May,[58] Ernst,[59] Woodhead and MacFadyean,[60] Martin,[61] Nocard,[62] Stein,[63] Hirschberger,[64] and others. Some observers, notably Martin[61] and Nocard,[62] found that milk was infective only when the udder was itself the seat of tuberculous lesions; but Ernst, as early as 1889, and Theobald Smith,[65] in 1893, from a number of experimental observations, concluded that tubercle bacilli may be present in the milk of tuberculous cows when the udder, so far as the naked eye could tell, contained no focus of disease. These conclusions are, moreover, supported by the investigations of Bang, Stein, Bollinger, Adami, Delépine, and Hirschberger.

Quite a number of observers failed to find tubercle bacilli in the milk of tuberculous animals; but May[58] and Hirschberger[64] proved that inoculation experiments are the more certain guide as to whether milk is infectious or not, since they obtained positive results from milk of undoubtedly tuberculous animals in which they were unable to demonstrate the presence of tubercle bacilli. As a matter of fact, Rabinowitsch and Kempener[66] obtained positive results

in inoculation experiments with the milk of ten out of fifteen cows that had reacted to tuberculin, and only two of these animals revealed udder lesions.

The general results of inoculation experiments differ in different countries, and naturally vary with the prevalence of bovine tuberculosis, the mode of technic, and many other factors.

The reports indicate that the infectious qualities are greatest with milk from animals with udder lesions, and next from those affected with general tuberculosis.

Bollinger[67] found that the general market milk of Munich proved infectious in 16.6 per cent. of the animals; Martin in Paris, in 33 per cent.; Bang in Copenhagen, in 15.5 per cent.; Ernst in Boston, in 12 per cent.; Fiorentini in Milan, in 10 per cent.; Rabinowitsch, in 28 per cent.; Obermüller, in 7.5 per cent.; Paris Municipal Laboratory, in 40 per cent.; Hamilton, Boyce, Woodhead, and Delépine in Liverpool, in 29 per cent.; Delépine in Manchester, in 25 per cent.; Massone, in 9 per cent.; Ott, in 11.6 per cent.; Jäger in Königsberg, in 7 per cent.; Kanthack and Sladen[68] found more than 50 per cent. of the milk supplied to the college in Cambridge infectious.

The experiments reported by Schmidt-Mühlheim, May, Stein, Hirschberger and Bang, and which were made with milk from tuberculous cows, proved successful in 60 to 70 per cent. of the animals.

In addition and, perhaps, of greater importance to the subject are, of course, the feeding experiments. Disregarding the earlier experiments of Gerlach and Bollinger, we have the important and positive results obtained by Baumgarten,[70] Wesener,[71] Fischer,[72] Peuch,[73] Ernst,[74] Bang,[55] Falk,[75] Zagari,[76] and Gebhardt,[77] showing that after a variable period, usually between eleven and twelve weeks, the ingestion of tuberculous milk developed a classical tuberculosis either of the intestinal mucosa and mesenteric glands, or spleen and liver, followed by subsequent involvement of other organs. In some, but not many, of these cases there was evidence of primary intestinal tuberculosis, but almost all of the cases revealed infection of the mesenteric glands. In the cases where the primary lesions were found elsewhere than in the intestines or mesenteric glands, Wesener,[71] in 1885, suggested that the bacilli must have found their way into the general circulation. As a matter of fact, the experiments of Dobroklonski,[78] Desoubry and Porcher,[79] of Nicolas and Descos,[80] have since shown that microbes may pass through the intestinal wall and enter the thoracic duct. The last-named experimenters, in 1902, brought out the interesting fact that three hours after the ingestion of tubercle bacilli the chyle and the lymph of the thoracic duct may contain bacilli in sufficient number to infect a guinea-pig, and we know from Wyssokowitschky's[81] experiments that from eight to thirty virulent tubercle

bacilli introduced into the peritoneal cavity of a guinea-pig are required to bring about infection.

It should be stated that in the feeding experiments by Günther and Harms,[83] Siedamgrotzky,[83] Roloff,[83] Bollinger,[42] and Nosotti the results were not uniformly successful, and that Schreiber,[85] Günther and Harms[82] have reported negative results.

On the whole, however, the feeding experiments with tuberculous milk proved infectious in about 45 to 50 per cent. of the cases. This seems excessive, probably because many of the negative results were never reported.

Apart from these experiments, we have the reports of Lucas and Morro,[86] Utz,[87] Klebs,[88] Kruckow,[89] Pfennigwerth,[90] Vollers,[91] Woodhead,[92] and others to show that the disease may be transmitted in milk to calves and pigs and other animals.

It is interesting to note that Bang,[55] May,[58] and Woodhead[60] noted a marked difference in the resulting lesions with raw milk and milk which had been subjected to different degrees of temperature. So, for example, Bang found that while a temperature of 158° F. did not destroy the vitality of the organisms, it reduced their virulence; and Woodhead, by the operation of certain low temperatures, obtained a diminution in the virulence of the tuberculous material in milk, which then "became so tardy in its operation on the test animals as to simulate the slower forms of consumption seen in the human subject, or when used to feed pigs—animals having some specialties of throat structure like that of man—gave rise to chronic enlargement of the throat glands resembling the scrofulous glands so common in children."

In the meantime 56 additional cases of transmission of bovine tuberculosis through the medium of the milk supply had been reported by Law,[93] Demme,[94] Sonntag,[69] Meyerhoff,[95] Hermsdorff,[96] Northrup,[97] Ollivier,[98] Wyss,[99] Gosse,[100] Rotch and de Schweinitz,[102] Law,[103] Rich,[104] J. L. Smith,[105] Ernst,[74] Brouardell,[106] Von Ruck,[107] Hills,[108] Prümers,[109] and Bovaird.[109] A number of accidental inoculations (wound infections) in man with bovine bacilli had also been placed on record by Pfeiffer,[110] Hartzell,[111] Rich,[104] Tscherning,[112] Law,[113] Ravenel,[114] Naughton[115]. These and the cases reported by Grothan,[116] Coppez,[117] and Priester[118] of primary subcutaneous tuberculosis caused by the topical application of cream, infection in milking, and the introduction of infected milk in the attempt to remove tattoo marks, had materially strengthened the evidence as to the transmissibility of bovine tuberculosis to man. Indeed, the absolute

identity of tubercle bacilli infecting mammalia was generally assumed until Theobald Smith,[119] in 1896, presented to the Association of American Physicians a comparative study of a bovine bacillus and a presumably human bacillus in which the morphological and biological differences were sufficiently marked to suggest further study; and as a result of this research Smith,[120] in 1898, stated that "we are justified if only to guide and stimulate further study in establishing a distinctively human or sputum and a bovine variety of the tubercle bacillus." He also expressed the opinion that the comparative study of the tubercle bacilli will lead to some definite understanding on certain important questions and eventually to more light on the whole subject of tuberculosis from the preventive as well as the therapeutic side. Notwithstanding these facts and the observations of Martin,[61] in 1895, that human sputum was far less virulent for animals than was bovine tuberculous material, the announcement of Koch before the International Congress for the Prevention of Tuberculosis, held in London July 22 to 26, 1901, was received with astonishment, especially since his conclusions carry with it a reversal of his judgment rendered in 1882.

Koch, in his address of 1901, makes the following declarations:[121]

"1. The bacilli found in cases of bovine tuberculosis are much more virulent for cattle and other domestic quadrupeds than the bacilli found in cases of human tuberculosis.

"2. This difference is so marked and so constant that it may be relied upon as a means of distinguishing the bacilli of bovine tuberculosis from those of the human disease, even assuming that the former may occasionally be found as a cause of disease in man.

"3. If bovine bacilli are capable of causing disease in man, there are abundant opportunities for the transference of the bacilli from the one species to the other, and cases of primary intestinal tuberculosis from the consumption of tuberculous milk ought to be of common occurrence. But post-mortem examination of human beings proves that cases of primary intestinal tuberculosis are extremely rare in man, and, therefore, it must be concluded that the human subject is immune against infection with the bovine bacilli, or is so slightly susceptible that it is not necessary to take any steps to counteract the risk of infection in this way."

Fortunately for the advancement of science, opposition fosters the spirit of inquiry, and many experiments have been made which have shed new light upon the already voluminous testimony. While it is true that the results in the feeding experiments with human sputum have varied, and that some of the earlier experimenters have reported negative results, the weight of the testimony is clearly in favor of the intercommunicability of human and bovine tuberculosis; and the experiments made during the past two years by Ravenel,[122] de Schweinitz,[123] Mohler,[123] Orth,[124] de Jong,[125] Stenström,[125] Fibiger,[125]

Wolff,[125] Nocard,[125] Arloing,[125] v. Behring,[125] Dean and Todd,[125] and Cipollina[125] conclusively demonstrate that cattle are not refractory to human tuberculosis and that bovine tuberculosis may be transmitted to monkeys. Whatever differences have been observed may very properly be attributed to the condition of the host and to the different degrees of virulence found in human bacilli, as shown by the work of Vagedes,[126] 1896, Lartigan,[127] and Delépine.[128]

The former studied twenty-eight human cultures, and found one much more virulent than the others and even more virulent than either of the two bovine cultures. Lartigan found bacilli of different degrees of virulence in human tuberculosis, and one of his specimens presented morphological and cultural peculiarities which suggested the bovine origin described by Smith. Delépine obtained from the human subject bacilli just as virulent as the bovine organisms, although in most instances the human tubercle bacillus was less virulent.

The writer has tabulated 86 cases of milk bovine tuberculosis, and quite a large number of cases of transmission of bovine tuberculosis to man have been reported by Krause,[128] Ravenel,[129] Lassar,[125] Hüls,[125] Spronck and Hoefnagel,[125] and Troje.[127] New light has also been shed upon the extent of intestinal infection by the statistics of Heller,[130] who finds that in 714 autopsies in children who died from diphtheria 140 (19.6 per cent.) were tuberculous, and of these 30.7 per cent. showed primary lesions in the intestines or mesenteric glands; 2 (1.43 per cent.) showed primary intestinal tuberculosis; 8 (5.7 per cent.) showed primary intestinal mesenteric glands; and 33 (23.5 per cent.) showed primary mesenteric gland tuberculosis.

Bovaird's[131] collection of primary intestinal tuberculosis in children is summarized as follows:

	Total.	*Intestinal.*	*Per cent.*
German	236	9	4
French	128	0	0
English	748	136	18
American	369	5	1

Dr. Bovaird confesses his inability to understand or explain the difference. The facts are that the post-mortem returns are extremely conflicting, and, unless accounted for by a variable prevalence of bovine tuberculosis and the habit of feeding raw or sterilized milk, suggest the necessity of more careful post-mortem work.

The statistical work by Gottstein[132] shows that the mortality from tuberculosis in breast-fed children is but slightly more than half as much as among those otherwise nourished, the proportion being as 6 : 10. Salmon[133] has analyzed the vital statistics of Massachusetts

and finds an increase of 36 per cent. in the forms of tuberculosis other than phthisis in the class under five years of age, while there was a reduction in the mortality of phthisis at all other ages of about 45 per cent., and the vital statistics of Michigan from 1885 to 1900 show also a tremendous increase during the milk-drinking age.

Dr. Hand's[136] statistics are as follows:

	Cases.	*Per cent.*
Total number of autopsies	332	
Total number showing tuberculosis	115	34.6
Apparent portal of infection (Hutinel's tuberculous chancre).		
Oldest lesion in the bronchial glands	75	62.2
Oldest lesion in the mesenteric glands	10	8.7
Process so general that the portal could not be determined	27	23.4

The cases of milk-borne tuberculosis, now numbering 86, while not as large as commonly supposed, are considerable when we consider the difficulty of tracing the infection on account of the more insidious character of the disease. In this connection we should remember that the first milk-typhoid epidemic was reported by Jacobs in 1858; in 1881 Mr. Ernst Hart had collected fifty epidemics, and in 1900 the writer tabulated 195 such epidemics. The opportunities for tracing bovine infection are much better in small communities and in isolated farm-houses than in large cities, and we should look to the country practitioner for help; he, in turn, unless properly equipped to verify his diagnosis, should apply to the Bureau of Animal Industry, or to the pathologist and bacteriologist of his medical school to check up his work.

A careful review of the cases recorded, when taken in connection with the laboratory experiments and the general evidence, justifies the following conclusions:

1. Tuberculosis may be transmitted to man in milk from tuberculous cows. The danger from this source is real and cannot be measured by the actual number of recorded cases, but should be judged in part, at least, by the inoculation and feeding experiments and the accidental wound infections which have established the intercommunicability of bovine and human tuberculosis.

2. The degree of danger may also be estimated by the prevalence of bovine tuberculosis and of the forms other than phthisis pulmonalis in man, remembering that the infectious qualities of milk are greatest when the udder is the seat of lesions, and that Gebhardt's experiments have shown that tuberculous milk when diluted with the milk of sound animals in the proportion of 1:40 lost its infective power.

3. The experimental studies also indicate that while the bacilli of human tuberculosis possess different degrees of pathogenic power and

are often of feeble virulence for cattle, Koch's assumption that human and bovine tuberculosis are distinct and that human tuberculosis cannot be conveyed to cattle appears to be disproved, and his failure to secure similar results may be attributed to the use of human bacilli of diminished virulence.

4. Recent investigations have strengthened Smith's claim that there are two types of tubercle bacilli—the so-called bovine and human types, possessing certain morphological and biological differences; but it has also been shown that virulent cultures may be obtained from both of these types, which when inoculated into animals produce the disease in question.

5. Further research seems desirable with a view of determining the frequency of primary intestinal and abdominal tuberculosis in all cases which come to autopsy, whether the child perished from tuberculosis or not; and in these autopsies the bacteriological examination should be directed to the existence of the two types of tubercle bacilli originally referred to by Smith, and whether the bovine type predominates in the so-called scrofulous lesions.

6. Careful chemical analyses of the milk of tuberculous animals should be made with a view of determining the amount of phosphoric acid as compared with the quantity in normal milk, since it appears probable from Dr. de Schweinitz's biochemical researches that the excess noted by the older chemists is really the result of bacterial activity in the udder of the cow.

7. In the meantime the pathologist has no occasion to reverse his opinion as to the identity of human and bovine tuberculosis, and the sanitarian has no reason to assume that the human subject is immune against infection with the bovine bacilli or is so slightly susceptible as to cause him to relax his efforts in preventive measures.

The writer desires to express his acknowledgments to Dr. D. E. Salmon, Chief of the Bureau of Animal Industry, U. S. Department of Agriculture, for material assistance in the preparation of this paper by placing at his disposal unpublished reports of experimental work.

Abstracts of Reports of Cases of Tuberculosis Attributed to Infected Cow's Milk.

Cases 1 to 16, 1846. Professor Klencke,[6] in a book of ninety pages, gives the clinical histories of sixteen children who had been fed with the milk of "scrofulous-tuberculous cows," and all of these cases point to tuberculosis of either the intestines, glands, skin, or bone. In three of the intestinal disorders he refers to the presence of indurated mesenteric glands. Of these cases one died, and two at the time of the report were in a hopeless condition. The clinical histories show that four of the children had been fed with milk from Cow B, three received their supply from Cow D, four from Cow E, two

from Cow F, and three from Cow G. Professor Klencke, having connected these cases with a particular milk supply, extended his investigation into the condition of the cows—their sanitary environments—together with a chemical and microscopic examination of the milk, and actually followed up his inquiry by four autopsies of the cows, in which he demonstrated scrofulous-tuberculous lesions. Such a course is worthy of emulation, and almost complies with the conditions of proof exacted by Professor Koch. Copious abstracts of these cases were published by the writer in the report of the Health Officer, District of Columbia, 1895.

Case 17. Schoengen,[45] at a meeting of the Veterinary Society held at Cologne, October 18, 1873, related the history of a child who, upon the advice of the family physician, was placed on cow's milk and developed undefined symptoms suggestive of tuberculous infection. An examination of the cow by a veterinarian resulted in a diagnosis of perlsucht, which was confirmed by post-mortem. A change of the milk resulted in subsequent improvement of the child.

Cases 18 and 19. K. Leonhardt,[46] December, 1874, reports the case of a child, previously healthy, which contracted tuberculosis soon after being weaned, and died. The autopsy revealed tuberculous meningitis and extensive lesions of the intestines and mesentric glands, which were undergoing caseous degeneration; attributed to the use of unboiled milk from a tuberculous cow.

The second case, also one of tuberculous meningitis, occurred in a previously healthy child after the consumption of milk from a cow which, upon examination, proved to be tuberculous.

He also mentions the history of a forester's family in which the children, although previously perfectly healthy, invariably developed tuberculosis after weaning. The cow was finally slaughtered, and the autopsy revealed extensive tuberculosis. A child, born subsequently, remained perfectly healthy.

Case 20. Professor O. Bollinger,[47] January 25, 1876, reports that Dr. Stang, of Amorbach, treated a boy, aged five years, well developed and of healthy parentage; no family history of tuberculosis for two generations. The child first developed symptoms of tabes mesenterica, and died some weeks later of miliary tuberculosis of the lungs, with enormous hypertrophy of the mesenteric glands, which were also undergoing caseous degeneration. While the post-mortem was being made it was ascertained that a short time previously the parents had a cow slaughtered, which was found by the veterinary surgeon at the abattoir to have phthisis pommelière. This cow had been a good milker, and for a long time the boy had been in the habit of drinking its milk, directly after the milking, in a raw state.

Case 21. Professor R. Demme,[48] 1879, reports the case of a previously healthy boy who was breast-fed until the fifth month, after which he was fed on cow's milk, and developed intestinal tuberculosis involving the mesenteric glands. The child died after three months' illness. Post-mortem examination revealed extensive tuberculosis of the intestines, especially of the jejunum and ileum and of the mesenteric glands; lungs and meninges showed no evidence of involvement. The cow died some months later, and Demme demonstrated, both macroscopically and microscopically, perlsucht.

Cases 22 to 25. Professor Demme, 1882, reports four cases of intestinal tuberculosis which, from the family history and the clinical and pathological evidence, he attributed to infection from milk from tuberculous cows.

Case 26. Alois Epstein,[49] 1879, reports the case of a child, three months old, in which the autopsy revealed follicular ulceration of the intestines, with involvement of the mesenteric glands and evidence of miliary tuberculosis in other organs. The author quotes Steiner and Neureiter, who in 302 tuberculous children found involvement of the lymphatic glands in 299 instances, and in 91 cases, or 30 per cent., the glands alone were involved. In 17 of 312 cases collected by Rilliet and Barthez there was no evidence of pulmonary lesions, and from this and a large amount of other evidence he

concludes that the portals of infection may be through the digestive as well as the respiratory tract.

Case 27. Professor J. Uffelmann,[50] 1880, reports the case of a child which was fed five months with milk from a certain cow. About the seventh month the child presented symptoms of bronchial catarrh and involvement of the superficial lymphatic glands. The child died at the age of eleven months with hectic fever and abdominal symptoms. No autopsy.

The cow was found to be tuberculous. The proof is not conclusive, but Professor Uffelmann is well known as a conservative writer and teacher.

Case 28. Albert Johne,[8] 1880, reports a case in which the post-mortem examination of a cow revealed extensive tuberculous deposits in the lungs and abdominal organs, which had been sent to him for inspection by the owner, who was especially interested in the result of the investigation, on account of the fact that up to within the past few weeks the cow was one of the best-nourished animals in his stables, and the milk had been selected by the steward for his infant son. The family physician was at once notified of the results of the autopsy, and reported progressive emaciation and a pulmonary catarrh which he had attributed to a recent attack of measles. A short time afterward Johne learned that the child perished from miliary tuberculosis of the brain.

Case 29. Law,[51] 1880, refers to a case reported to him by Dr. Corliss, of New Jersey. A family cow, supposed to be suffering from the lung plague, was found to be affected with tuberculosis instead, and the owner's wife (a consumptive), who had been making free use of the milk, warm from the cow, was persuaded to give it up and underwent decided improvement.

Cases 30 to 33. Law,[93] 1883, cites two cases, daughters of a Scotch family, strong and healthy, who were brought up on milk of tuberculous cows and died of tuberculosis, while two sons in the same family who did not use the milk remained healthy. See, also, Hills and Rich in *Bulletin* 42, *Vermont Vet. Exper. Station*, p. 55.

Mr. Howe, of North Hadley, Mass., lost a son, twenty months old, from abdominal tuberculosis three months after he paid a week's visit to his uncle and had been fed with the milk of the uncle's cow. The cow was killed soon after and proved to have generalized tuberculosis. The child's sickness and wasting began a few weeks after he returned home. He had previously been strong and healthy, as were and are his parents. (Cited by Law from *Report of Vet. Congress*, at Brussels, 1883, p. 288; also, by Nocard from *Compte rendu of Fourth Internat. Vet. Congress*, Brussels, 1883, p. 188; and by Salmon, *Bulletin No.* 33, *Bureau of Animal Industry*, 1901, p. 21.)

Case 34. H. Meyerhoff,[95] 1884, reports the case of a child, seven years of age, of healthy parentage and with a good family history, who developed tuberculosis after drinking for a long time milk from a cow suffering with pearl disease. Although the patient had also worn some infected clothing, Meyerhoff attributes the infection to the milk.

Case 35. Demme,[48] 1886, reports the case of a boy, four months of age, who developed tuberculosis of the mesenteric glands without the slightest lesion in the intestinal walls. The child was fed on unboiled milk from a tuberculous cow, as shown by the autopsy. Tubercle bacilli were isolated from the udder as well as from the mesenteric glands.

Among 2000 tuberculous children treated during the past twenty years at the hospital Dr. Demme, a very conservative and competent authority, reported this and the preceding cases as milk-borne infections.

Case 36. Law,[93] 1894. A child, four years old, son of Col. Beecher and a grandson of Henry Ward Beecher, died March, 1894, at Yonkers, N. Y., of tuberculous meningitis. The diagnosis was confirmed by specialists. There were no known hereditary tendencies to the disease. The certainty that he had the disease, and the inability to account for it from human agencies, led the physician to suspect the milk of two Alderney cows, on which the child had been mainly fed. The tuberculin test and post-mortem showed that both animals were tuberculous.

Case 37. Sonntag, quoted by Baum,[95] page 196, reports the case of a child, aged six months, which died from tuberculous meningitis. The parents were perfectly healthy, but the child had been almost exclusively fed with milk from a tuberculous cow.

Case 38. Hermsdorf[96] reports the case of a girl, aged fourteen years, of good family history and healthy stock, who died from tuberculous laryngitis. The most extensive lesions were found in the ileum and cæcum. The pulmonary lesions were slight. Inquiry revealed the fact that the girl had been in the habit of drinking the milk while still warm from a tuberculous cow.

Case 39. W. P. Northrup,[97] 1890, reports the case of an infant, aged fifteen months, which was brought to the New York Foundling Hospital when two hours old, placed with a wet-nurse, from whom it was taken in the twelfth month on account of its miserable condition, developed general tuberculosis, —primary intestinal infection—possibly from food infection.

Cases 40 to 52. Ollivier,[98] 1897, presented to the Paris Academy of Medicine, February 24, 1891, the history of twelve young ladies, scholars in the Pension of the Dames Blanches at Chartres, all suffering from tuberculosis, of whom five died, the last death from tuberculous meningitis. A few days later the milch cow was butchered and found to be suffering from tuberculosis of the udder and of the internal organs. In almost all of the cases there was evidence of intestinal involvement, and in none of the cases was there an inherited predisposition. Professor Koch in a recent address states that Ollivier in a subsequent communication before the Academy remarked that only the servants had consumed the milk from the tuberculous cow.

Cases 53 and 54. Professor Oscar Wyss,[99] 1893, reports three cases in 123 autopsies on children, aged five and three-fourths years, six and a-half years, and two years, coming within the observation of competent clinicians during a period of three years, in which tubercle bacilli were found in the primary intestinal lesions, and all were strongly suspected of being caused by infected cow's milk.

Case 55. Gosse,[100] 1893, reports the case of his own daughter, who, up to within a year of her death, was perfectly healthy, developed tuberculosis of the intestines and mesentery, which the father traced to the consumption of raw milk from some of the cows on his own farm, which were found to react to tuberculin, and post-mortem examination revealed tuberculous lesions of the udder.

Case 56. Thomas M. Rotch and E. A. de Schweinitz,[102] 1893 or 1894. The case was under the care of Dr. Rotch in the Children's Hospital in Boston in 1893 or 1894. The particulars were sent to Dr. de Schweinitz, of the Bureau of Animal Industry, by Dr. Rotch. A boy, aged seven years, had been drinking a great deal of milk from one cow for about four months before entering the hospital. The cow was found to be tuberculous, the disease being demonstrated by autopsy. The boy was a case of primary tuberculous peritonitis with effusion. Laparotomy was performed by Dr. Bradford and a large amount of serous fluid evacuated. The peritoneum was found to be thickly studded with minute tubercles, and the presence of tubercle bacilli was demonstrated. Four years after the operation the boy was found to be perfectly well, and seemed free from tuberculosis.

Cases 57 to 64. Brouardel,[106] 1894 (quoted by Harrington, *Practical Hygiene*, 1902, page 102), records the death of seven children with no hereditary taint, inmates of a convent, from tuberculosis, supposedly induced by the use of milk from a cow with tuberculosis of the udder. Another case reported by him and quoted by Freudenreich (*Les Microbes es leur des Role dans la Laiterie*, Paris, 1894. p. 45) is one in which five of fourteen girls in a boarding school became infected and died. The milk which they had used daily came from a tuberculous cow. These cases and Ollivier's collection are of special value on account of the number of persons affected who have taken the same milk, the intestinal lesions, and the demonstration of tuberculous udders.

Case 65. Law,[103] 1894, reports the case of a strong, vigorous boy, aged one and a-half years, who for a week drank the milk of a cow which was

shortly afterward condemned and killed in a state of generalized tuberculosis. In six weeks the child was noticeably falling off, and in three months died, a mere skeleton, with tuberculosis of the abdomen. The father could trace no tuberculosis in his near ancestors, but the mother's father and uncle had died of it. She, herself, was in perfect health.

Cases 66 to 68. F. A. Rich,[104] 1895, reports several cases: Case 1, Mr. C., a young man of healthy parents, died of tuberculosis. Three years later Rich tested the herd of cows from which the patient had used milk plentifully, destroyed sixty-five out of seventy-four, many of which proved, upon post-mortem examination, to be affected with general and far-advanced tuberculosis. A few months later the owner of the herd developed acute pulmonary tuberculosis, and at the close of the report was in an advanced stage of the disease. Case 2, Mr. D., a young man, died of pulmonary tuberculosis. Two months later Rich tested the herd and destroyed eighty animals, nearly 90 per cent. of the herd. Several calves and hogs were found to be tuberculous from feeding upon the milk. Case 3, Miss R , using milk from a tuberculous cow, developed symptoms of pulmonary consumption. The cow was exchanged for another, and she apparently recovered. Three years later she went to live where the above-mentioned cow was still owned and milked, and in a short time developed acute miliary tuberculosis, from which she died a month later, and the autopsy revealed general tuberculosis.

Case 69. J. L. Smith,[105] November 14, 1895, reports a case of tuberculosis in which it seemed clear that the disease had been transmitted to the little one by infected milk. The child was in charge of one of the wet-nurses of the New York Foundling Asylum, and then had been partly fed on milk from a small grocery store. Dr. R. G. Freeman found no ulceration of the intestines. The condition of the mesenteric gland, that had undergone cheesy degeneration, showed that here was the oldest lesion, and it, therefore, indicated an intestinal infection.

Cases 70 to 81. H. C. Ernst,[59] 1895. Of the 1013 replies from physicians to Ernst's questions whether they knew of cases of transmission, 895 were in the negative; 8 reported transmission from mother to child; 11 reported cases of infection through cow's milk, and 16 reported suspicious cases. Of 54 replies to the same question from veterinarians, 14 reported positive cases and 9 suspicious cases.

Case 82. M. Volkoff,[109] 1895 ("Primäre Tuberkulose des Verdauungstractus," *Gazetta Bolknia*, 1895, No. 51, quoted in Lubarsch u. Ostertag's Ergeb. der Allg. Pathologie, 1899, p. 369), reports a case of primary tuberculosis of the intestinal tract, localized in the jejunum; from there it extended to the neighboring retroperitoneal and mesenteric glands and spleen. All other portions of the intestinal tract were normal.

Cases 83 and 84. Von Ruck, 1899 (quoted in Dr. Salmon's Bulletin No. 33), reports two cases, father and child, which he considered almost conclusive. The former developed a case of acute miliary tuberculosis. The autopsy revealed no old focus in the chest or abdomen. The child, aged nearly one year, was first treated for fever and diarrhœa, but later developed symptoms of meningitis, and died. The milk which the child had received came from a Jersey cow, and no other had been used up to the time the diet was changed on account of the diarrhœal attack. The father had drunk from a pint to a quart twice a day, warm, at the time of milking, of this cow's milk. Upon post-mortem examination of the cow there was found extensive tuberculosis in the lungs and peritoneal cavity, and the lump in the udder contained a caseous centre, which, on examination, showed numerous well-stained tubercle bacilli.

Case 85. David Bovaird, Jr.,[109] 1891 ("A Case of Primary Intestinal Tuberculosis," *Archiv. of Pediatrics*, 1901, vol. xviii. p. 894), reports a case, aged three years, a patient of the New York Foundling Hospital. Family history, nothing known. No acute illness, but boy had always been weak and sickly. He was cared for outside the institution until August, 1899. The diagnosis of primary intestinal tuberculosis is based upon the autopsy findings. The oldest and most advanced lesions were in the mesenteric node.

The conclusion that the intestine has been the entry-port of the infection, Dr. Bovaird thinks can hardly be questioned. It was not possible to investigate the milk supply, but the case seemed to Dr. Bovaird to deserve to be classed as one of "Fütterungs-tuberculose."

Case 86. Ebers[109] (quoted by Bovaird) reports six cases in which tuberculosis in children was attributed to the consumption of the milk of tuberculous cows. Four of these, collected by Baum, were reported by Hermsdorf, Leonhardt and Sonntag. The remaining two were reported by Johne and Prümer. All but Prümer's case have already been presented in this table.

S. Delépine, 1898 ("A Lecture on Tuberculosis and the Milk Supply," *Lancet*, London, 1898, vol. ii. pp. 733–738), refers to Dr. Simcock's work in his laboratory, who selected thirteen cases of wasting in children, due to improper feeding, out of many more in which there was no evidence of tuberculosis in the parents or in the various organs of the children as examined in the post-mortem room. Dr. Simcock considered the cases free from tuberculosis, but Delépine was not quite convinced. On close examination of the mesenteric glands, two presented clear tuberculous lesions under the microscope and five more suggested the possibility of incipient tuberculosis.

Accidental Inoculation of Man with Bovine Bacilli (by Topical Application of Cream and Milk).

Grothan,[116] 1896, reports the case of a little girl, aged six years, suffering from an eruption on the left leg, supposed to be due to ivy poisoning. This was treated at home by the topical application of fresh cream. When seen by Grothan he recognized the characteristic appearance of a tuberculous ulcer. The cow was examined and the udder seemed normal, yet inguinal and intraperitoneal inoculation of two rabbits with a mixture of milk and cream gave positive results. The caseous material from the nodules of the girl's leg injected into the peritoneum of a rabbit produced death from tuberculous peritonitis.

Coppez,[117] 1896, reports the case of a girl, aged seventeen years, who had a wound on the palmar aspect of the third finger, which became infected with tubercle bacilli during milking. The original lesions gave rise to over thirty-five subcutaneous abscesses in different parts of the body within six months, and subsequently more appeared, in all sixty to sixty-six. At no time could any visceral lesions be recognized, the lymph nodes of the neck being greatly enlarged; after several months death ensued.

Priester,[118] 1896, p. 11, reports the case of a young man who tried to remove a number of tattooes from his hands and forearms by introducing milk into the tattooed parts by means of needle punctures. Later there appeared on the back of both hands at points which had been so treated bright red spots the size of millet seeds, containing a yellow center. Pus was evacuated from several nodules. These were found to be lupus nodules in which giant cells were demonstrated. The diseased parts were cut out and the wounds healed without a recurrence of the trouble.

Wound Infections.

L. Pfeiffer,[110] 1885, attended, at Weimar, a veterinarian, aged thirty-four years, of good constitution and without hereditary predisposition, who, in 1885, pricked his left thumb in making an autopsy on a tuberculous cow. The wound healed, but six months later the cicatrix still remained swollen, and in the autumn of 1886 the man had pulmonary tuberculosis, with bacilli in his sputa, and death occurred in two and a half years after the wound. Post-mortem revealed tuberculosis of the joint of the wounded thumb, and in the lungs extensive tubercles and vomicæ. The joint lesions showed caseous masses extraordinarily rich in tubercle bacilli.

Tscherning,[112] of Copenhagen, 1888 (*Proceedings of Congress for the Study of Human and Animal Tuberculosis*, first session, Paris, 1888, p. 275. Cited by Law, Bujwid,[119] and Salmon), attended a veterinarian who had cut

his finger in making an autopsy on a tuberculous cow. The wound healed, but there remained a swelling which soon ulcerated and refused to heal, so that the whole tumefied mass had to be cut out. The microscope revealed a distinct tuberculous process and the presence of the characteristically staining bacilli. The patient recovered without further development of the disease.

T. A. Rich,[104] 1895. (*Veterinary Magazine*, December, 1895, vol. xi. p. 729.) Mr. T. cut his finger on a spicula of bone while making post-mortem examination of tuberculous cows. In a few weeks he developed a tuberculous joint and a few months later showed unmistakable signs of phthisis.

M. B. Hartzell,[111] Philadelphia (American Medical Association, June 1–4, 1897), reports a case of local infection in the back of the hand in the person of a previously healthy man employed to clean cars used for the transportation of cattle; the wound was caused by a piece of broken woodwork of a car, producing a typical verrucous tuberculosis, which appeared to yield to treatment. Within a year the patient developed pulmonary tuberculosis, and finally died from general tuberculosis.

M. P. Ravenel,[114] 1897, reports the case of a veterinarian who cut the knuckle of his forefinger while making a post-mortem examination of a tuberculous cow. The wound healed badly, remained swollen, and showed a decided tendency to ulcerate. Removal of the cicatricial mass was practised and the tissues sent to Dr. Ravenel for examination. They showed typical tuberculous lesions with giant-cell formation.

M. T. Naughton,[115] of Chicago, Illinois, 1899 (cited by Dr. Salmon, *Bulletin* No. 33, p. 19), reports the case of a man, aged thirty-four years, with a good family history, a butcher by occupation, who on May 3, 1899, while cleaning cattle viscera, fell, and a meat hook upon which hearts and lungs are hung penetrated the right hand between the second and third metacarpal bones. A tendovaginitis with some lymphangitis of the arm resulted; made a good recovery; four months afterward an abscess formed in the axilla, which was cleaned, and tubercle bacilli were demonstrated in the broken-down gland tissues. In three months afterward, or seven months from the original accident, he died from pulmonary tuberculosis.

M. P. Ravenel,[114] 1900, reports three cases of veterinarians in the State of Pennsylvania, of local infection from wounds in the finger inoculated accidentally with bovine tuberculosis in performing autopsies. In the first case a large giant cell but no tubercle bacilli were demonstrated in the excised portion. In the second case two guinea-pigs inoculated with a portion of the excised nodule developed a generalized tuberculosis, and in the third case Dr. John Guitéras demonstrated tubercle bacilli in the excised nodule.

Law,[113] 1901 (cited by J. J. Repp, *American Veterinary Review*, New York, 1901 and 1902, vol. xxv. pp. 624–636), reports that a young veterinary friend, who was inoculated in the hand in opening a tuberculous cow, suffered from a tumefaction of the resulting cicatrix in which tubercle bacilli were demonstrated.

M. P. Ravenel[129] reports the case of his assistant, who was inoculated with bovine tuberculosis, and from whose tissues tubercle bacilli were isolated after fifty-eight days.

C. H. H. Spronk[133] and K. Hoefnagel, 1900 and 1901 ("Transmission a l'homme, par inoculation accidentelle de la tuberculose bovine, et réinoculation experimentale au veau," *La Semaine méd.*, Paris, Ann. 22, No. 42, October 15, pp. 341–343; abstracted from *American Medicine*, November 8, 1902, p. 749), report the case of a butcher accidentally wounded, May, 1900, in the finger while assisting in inspecting the tuberculous organs of a cow. The wound healed promptly, but later the finger presented evidence of infection. In February, 1902, Professor Nareth extirpated the affected portion of the skin together with the tumefied cubital ganglia. The tuberculous character of the lesions was demonstrated by microscopic examination and by the inoculation of guinea-pigs, and an emulsion made from the spleen of one of these pigs was used to inoculate a calf previously tested with tuberculin, and the animal placed in a newly-constructed stable, with the result of producing

general tuberculosis as demonstrated by the presence of giant cells and numerous tubercle bacilli. Finally two guinea-pigs which had been inoculated with material obtained from this calf died of tuberculosis.

Lassar[133] ("Ueber Impfluberkulose," *Deutsche med. Wochenschrift*, No. 40, October 2, 1902, pp. 716–718) reports thirty-four cases of verrucose tuberculosis following wounds in 108,000 patients, but only four of these cases were butchers. Later he examined 365 men employed in abattoirs and found seven suffering from inoculated tuberculosis, while three others he considered as possibly affected. (This shows a percentage of two to three among men employed in the abattoirs, and only one-third of one per thousand among persons otherwise employed.)

Hüls[133] ("Zur Frage der Uebertragung der Rindertuberkulose auf den Menschen, *Münch. med. Wochenschrift*, No. 24, June 17, 1902, pp. 1003 and 1004) reports the case of a wealthy miller's family, composed of the wife, five sons, and two daughters, herculean in stature, and boastful of strength and health. Consumption had never occurred in the family of either parent. Of these, all but two died from consumption within a few years, and even one of the surviving sons was treated for a tuberculous abscess of the finger. Hüls states that all these cases appeared subsequently to the introduction by the miller of a herd of Simmerthal cattle on his farm, which were nearly all infected with tuberculosis.

Krause[133] (quoted by Salmon) reports a case of a butcher, aged thirty years, with a good family history, whose duty it was to remove diseased parts of cattle killed for food. The infection was located in the right arm, and was attributed by the man to a splinter of wood run into his right thumb three years ago. Immediately after the accident he had removed the hide from a sick cow. Pieces of gland and skin from the arm proved on examination to be tuberculous.

Troje,[137] 1903, reports the case of a young, healthy male, who gave no personal or hereditary history of tuberculosis. He was inoculated with bovine tuberculosis on the hand, and a typical lupus lesion developed on the site of inoculation. Subsequently the lymphatic glands which drain the involved region became tuberculous, and in the course of time many other lymphatic glands throughout the body became similarly affected.

Recent Researches with Reference to the Communicability of Human Tuberculosis to Animals.

Chaveau's experiments (quoted by Salmon[2]) show that cattle were successfully infected with human tuberculous material by ingestion, intravenous injection, and by subcutaneous inoculation.

Sidney Martin,[133] 1895 (*Report of Royal Commission Appointed to Inquire into the Effect of Food Derived from Tuberculous Animals on Human Health*, London, Part I., 1895, pp. 9–11), also obtained successful results with bovine animals by feeding sputum.

Thomassen[133] ("The Receptivity of Bovine Animals for the Bacillus of Human Tuberculosis," *Transactions of the British Congress on Tuberculosis for Prevention of Consumption*, London, July 22–26, 1901, vol. iv. pp. 21–27) reports an experiment in which a calf was inoculated in the anterior chamber of the eye with a pure culture of tubercle bacillus isolated from a case of tuberculous arthritis in man. When killed, after six weeks, both lungs were found to contain numerous miliary tubercles and some gray fibrous tubercles of larger size. The path of infection from the eye could be traced by the lymphatic glands.

Nocard,[133] 1901 (*Press. Vét.*, Angers, vol. xxi., November 30, 1901, pp. 398–403), states that cattle are not refractory to human tuberculosis.

S. Arlong,[133] 1901 ("L'inoculabilité de la tuberculose humaine et des idées de M. Robert Koch sur cette tuberculose et la tuberculose animale," *Bull. acad. de méd.*, Paris, vol. lxv., 3 ser., xlvi., No. 43, December 24, 1901, pp. 897–911), reports experiments with three different cultures of human origin. One of these had been in his laboratory since 1896. It was capable of in-

fecting guinea-pigs and rabbits by subcutaneous inoculations; and intravenous injection with an emulsion of this culture in a heifer calf, two sheep, and a kid conveyed the disease, as the autopsy demonstrated tuberculous lesions in all these animals.

M. P. Ravenel[122] reports having fed four calves with human sputum, and post-mortem examination proved that all had become affected with tuberculosis, the lesions in two being quite extensive. His inoculation experiments in calves and cows with cultures obtained from the mesenteric glands of a child which died of tuberculous meningitis proved very virulent for bovine animals.

De Jong[123] ("Experiences comparatives sur l'áction pathogène pour les animaux, notamment pour ceux dé léspèce bovine, des bacilles tuberculeux provenant du boeuf et de l'homme") reports a series of inoculations in seven bovine animals (calves and steers); all became tuberculous by the injection of tubercle bacilli of human origin. In one of the animals the disease was serious and extended; in four others it had a retrogressive tendency, and in two it was progressive. He concludes that bacilli isolated from the human body or from sputum are capable of causing tuberculosis in cattle.

J. Orth[130] ("Ueber einige Zeit-Streitfragen aus dem Gebiets der Tuberculose," *Berl. klin. Woch.*, August 25, 1902, pp. 793–978) showed by inoculation experiments that a progressive fatal tuberculosis was produced in the animals by material originating in the human subject.

Olof Stenström[133] ("Die Tuberculose des Menschen u. der Rinder," *Zeitschrift f. Thiermed.*, Band. vi. and vii., Heft 4, 1902, pp. 289–291), from his experiments, states that he found it quite easy to infect cattle with human tuberculosis.

Max Wolff,[133] 1902 ("Perlsucht und menschl. Tuberkulose," *Deutsche med. Woch.*, 28 Jahr., No. 32, August 7, 1902, pp. 566–570), reports inoculation from cases of primary tuberculosis of the intestines in man, from which he concludes that pearl disease of cattle may appear in man.

Johannes Fibiger and C. O. Jensen,[133] 1902 ("Uebertragung der Tuberculose des Menschen auf das Rinder," *Berl. klin. Woch.*, 39 Jahr., No. 38, September 22, 1902, pp. 881–886), conclude by stating that Koch announced that one could distinguish between human and bovine tuberculosis by inoculating calves subcutaneously. If this is true, then they are of the opinion that three of the calves cited in their experiments must be considered as perlsucht, and the idea that tuberculosis of cattle is not virulent for man is disapproved.

Dean and Todd,[133] 1902 ("Abstract of Certain Experiments on Tuberculosis," *Lancet*, London, November 1, 1902, pp. 1186, 1187), from their experiments conclude that the human tubercle bacillus is by no means innocuous to the calf, as the control animal injected directly with sputum contracted an extensive glandular tuberculosis.

E. v. Behring,[133] P. Romer and G. Ruppel (*Tuberculose*, xviii. p. 90, Marburg, 1902) report experiments in which human sputum was used to inoculate a guinea-pig, and a culture from this animal's spleen was used for inoculating a goat, and an emulsion of the spleen of the goat was passed through a series of guinea-pigs, and a culture obtained from the spleen of the third guinea-pig was inoculated into a calf by intravenous injections. The calf died after four weeks directly from the tuberculosis thus produced. A cow was also inoculated November 26, 1901, with a tuberculous culture of human origin after a single passage through a guinea-pig, and on January 14, 1900, the affected eye was enucleated and a pure culture was obtained from it after one passage through a guinea-pig. The cow at the close of the report was gradually losing ground.

De Schweinitz's[133] recent experiments with intravenous injections and inoculations of cultures obtained from the mesenteric glands of children affected with tuberculous peritonitis produced fatal generalized tuberculosis in calves, and a steer inoculated subcutaneously with a piece of tuberculous tissue from one of the children also produced tuberculosis.

Mohler's[133] experiments also show "that it is not a difficult matter to

obtain very virulent tubercle bacilli from human sources, and that some of these are just as virulent as bovine bacilli."

A. Cipollina[135] reports that a monkey fed with bovine milk containing tubercle bacilli cultures contracted tuberculosis, with no primary lesions apparent in the gastrointestinal tract. On the other hand, inoculation of a calf with human bacilli resulted negatively.

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TABES IN THE NEGRO.*

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About the negro much has been set down in the annals of anthropology, ethnology, and sociology, and a glance at medical literature as it appears in summary form from year to year would suggest that almost the last word has been written about tabes; but of tabes in the negro I have been able to obtain only the fewest references recorded, in the main, by American writers. Von Leyden[1] quotes Buri as sponsor for the statement, "*bei den so häufig syphilitischen Negern soll die Tabes sehr selten vorkommen*" (tabes occurs very rarely in the large number of syphilitic negroes). In another foreign contribution to the literature of this subject, entitled "*La Ataxia Locomotriz (en el negro),*"[2] is recorded the fact of tabes occurring eight times in full-blooded negroes and three times in mulattoes.

C. W. Burr[3] in a report on "The Frequency of Locomotor Ataxia in Negroes," read before the Philadelphia Neurological Society, 1892, prefaces the comments on his own case with the written opinions of many Southern practitioners, all of whom were unanimous in declaring tabes in the negro of rare occurrence. Professor Osler is also quoted, in answer to Dr. Burr's query, as stating "of twenty cases under

* Read at the meeting of the Chicago Neurological Society, October, 1903.

observation since the opening of Johns Hopkins Hospital, one was a full-blooded negro and one a mulatto." Dr. Potts, physician to the Nervous Dispensary of the University of Pennsylvania, told Dr. Burr that he had not seen a case in eight years. During fourteen years, says Dr. Burr, not a case applied at the Infirmary for Nervous Diseases in Philadelphia. He adds: "For my own part, I have never seen a case in the pure black, and but one in a mulatto." McConnell[4] presented a report of five cases before the Philadelphia Neurological Society in 1899, in which he stated that he had seen "tabes in many whites, a few cases among the mixed races, and but five instances in *pure* negroes." Four of these cases were in males and one in a female. Dr. J. H. Lloyd,[5] in 1896, reported one case in a negress. In a tabulation of 194 cases of tabes, with a view to demonstrating the distribution among the different nationalities, F. Savary Pearce[6] gives the following:

American and English	119
French	2
German	21
Hebrew	6
Irish	25
Colored (not pure)	2

The next reference in American literature, and one of more recent date, is that of A. P. Francine,[7] who discusses briefly "Two Cases of Tabes Dorsalis in Negroes—Husband and Wife." In this article S. Weir Mitchell is credited with saying that he has seen four cases of tabes, two of which had occurred in mulattoes in Florida.

Aside from these allusions in the literature to the subject-matter of this paper, it has become, I believe, almost a habit with clinicians to regard the colored people as exempt from this disease. Neurologists as well, who from year to year attract an ever-increasing, vast clinical material, confess to having seen little or no tabes in the negro. My chiefs in the Department of Nervous Diseases at the Northwestern University Medical School, Professors Hugh T. Patrick and Archibald Church, record having met with three cases each in their entire experience.

It has been my good fortune during the past year to see and study four undoubted cases of tabes in colored individuals, three of them being in females and one in a male. Although adjudged in the light of sheer coincidence, this ratio of three to one in favor of the female is unusual, since statistical plurality is quite the other way. The clinical picture in these cases in no way deflects from the symptom-complex we are accustomed to see in the whites except, perhaps, in the fact that they are all of the "amaurotic type" of the disease. Satisfied on the score that there is nothing of a pathological nature to reveal, I am, nevertheless, mindful of other points of interest which I believe should

command the attention of medical men, and neurologists in particular, and for the following reasons:

1. The relative vital movement of different races in a mixed population is a matter of interest to the physician as well as the ethnologist.

2. The white and colored races living side by side in this country and in some of the Southern States in nearly equal numbers afford an opportunity for study, though the problem is rendered complicated yet interesting by their partial admixture.

3. The compilations and statistics in the past on "race and disease" have slighted certain diseases of which we are in duty bound to know more than we do, in order to avert inadequate and incomprehensive conclusions.

4. Tabes in the pure and hybrid negro should afford fresh incentive for a study and investigation of the, in large part accepted, in part conflicting, and in part rejected, evidence of a luetic etiology.

5. The influence of race amalgamation on production and inhibition of disease (with special reference to civilization and syphilization as a factor in the production of tabes) is important in all lands.

To quote from that brilliant and exhaustive treatise, "The Surgical Peculiarities of the Negro," written by Dr. Rudolph Matas,[8] of New Orleans, and delivered at the 1896 meeting of the American Surgical Association, "the influence of crossing and a comparative study of the negro is a prerequisite to the understanding of medical shades of difference."

These questions can only be approached by a better understanding of the vicissitudes through which the negro has passed since the days of his importation from Africa, but an historical commentary treating of the native African negro would far exceed the intended scope of this paper. Concerning negroid ancestry in Africa, I might say that valuable accounts of the endemic diseases afflicting the negroes who live in semisavage or tribal state in Africa appeared in 1895 in communications to the German Government by Dr. A. Plehn.[9] He dwells at length upon all the diseases to which the negro is prone, briefly classifying the nervous diseases as follows:

"Beriberi prevails in Kamerun, but perhaps to a greater degree in Sierra Leone and Congo. I have seen a single case of hysteria (*einen ausgesprochenen Fall*), with all its signs and symptoms, in the wife of King Bell. Epilepsy occurs; I recall four cases (*ausgeprägte Fälle*) in Kamerun." This exhausts his list.

When the negro was first imported he brought with him diseases peculiar to himself (*sui generis*) and foreign to the inhabitants of our colonies; yaws—the sleeping sickness—and elephantiasis belonged prominently to the earlier era in this country. A study of these and other diseases was immediately begun by Southern practitioners, and

the numerous authentic works extant as classics bear witness to the untiring effort and careful observation made by the Southern profession. The names of Nott (physician and anthropologist), Gliddon, Cartwright, Grier, Corson, and Tipton are inseparably linked with the literature exploited in the Southern medical journals as far back as 1850. Trismus nascentium—marasmus—referred to by Grier as "literally a wasting away—a tabes of young life—provincially styled 'the drooping disease of negro children,'" invermination—cachexia Africana—(dirt-eating disease of the negro) are familiar topics in the pioneer literature of the South. Grier,[10] in 1852, said: "Whether the races be derived from one or more original species, they are now characterized by such peculiarities of constitution and temperament as to make the distinctive maladies of the negro race a subject of much practical importance to us and worthy of a more painstaking investigation than it has ever yet received from the hands of the medical profession of the South."

According to the consensus of medical and statistical opinion, release from bondage has been the turning-point in the negro's physical welfare. Such medical writers as Tipton, Furniss, and principally Corson and Matas, pronounce this as the beginning of his downfall, his physical deterioration and moral degradation. Since the war and emancipation the avenues of independent occupation have been flung wide open to irresponsible beings, unprepared for their unequal lot. The introduction of civilized vices into uncivilized communities began anew, and the negro was quick to accept and devour the vices and excesses of material civilization. Out of the general morbidity and depravity of this new community of interests sprang a mortality record which was immediately higher than that of the whites in ante-emancipatory times and has been getting increasingly greater in later years, until now the nation feels a keen concern in the gravity of the "vital statistics of the negro."

Diseases incident to the state of slavery are disappearing, and tuberculosis, syphilis, and cancer are deep-rooted in the colored people. Old immunities are lost. Admixture with white blood has weakened the race. In the presence of new blood has come the tendency to acquire new diseases; the amalgamation is responsible for much that is radically new in the pathological field.

By a legitimate intermingling of tribal strains of blood originally distinct in Africa and an illegitimate, universal miscegenation with the whites, the typical American negro of to-day has incorporated in his formation certain generic characteristics of his negroid ancestry plus the deteriorating phenomena of his hybridity.

Case Histories. Whereas, the cases I am about to report do not present in number or variety the symptoms we are accustomed to asso-

ciate with this essentially chronic disease of the nervous system as it occurs in the whites, nevertheless, the clinical syndrome in each case precludes the serious consideration of any other disease. The cases tabulated here were seen in Dr. H. T. Patrick's clinic, Northwestern University Medical School.

CASE I. (Referred from Eye Department, Northwestern University Medical School, by the courtesy of Dr. Richard S. Patillo, who anticipated the possible diagnosis of tabes fom the "fundus" findings.)—February 4, 1903, M. R., aged thirty-nine years, female, colored, married. Born in Louisville, Ky., of black father (slave) and bronze mother (slave). The father lived to grow very old at the Soldiers' Home, Wyandotte, Kansas; cause of his death unknown. Mother lived to the age of sixty-five years and died of some asthmatic trouble.

The patient, as a young girl of twelve years and upward, was subjected to exposures of wet and cold, which led to the development of rheumatic symptoms that have prevailed from year to year with moderate severity. The hardest of work was required of her in an attempt to support the family. One brother died of smallpox at the age of twenty-three years, and one sister died of pneumonia at eighteen years. At the age of sixteen years the patient miscarried an illegitimate child at four months. She attributes this occurrence to a fall which she experienced at the time. At eighteen years she married the father of her first illegitimate, and gave birth to a full-term, living male child in the first year of her marriage. Prior to the birth of the child she records having had a protracted siege of sore throat, and adds that the husband abandoned her, with the excuse that he was afraid of giving her some "bad disease." She denies knowledge of specific taint, and upon inquiry records no signs of secondaries. I detail the data of ancestry and venereal infection, holding them to be particularly important in the anamnesis of these cases. At the age of eighteen years she had what she pleases to call an attack of pneumonia, but since that time and up to the date of onset of the present trouble she has had "no misery nor corruption anywhere."

Present Illness. Onset about five years ago, with severe headaches persisting for a week at a time, disappearing and recurring for longer or shorter intervals. Headaches agonizing, occipital, vesperal. Simultaneously, dimness of vision in left eye, which glasses did not seem to relieve. Failing vision progressive until two months ago, when she noticed complete blindness in the left eye. Sight in the right eye began to fail shortly before the vision in the left eye was destroyed, and is becoming more and more impaired. Early in the course of the trouble patient experienced severe paroxysmal pains in the stomach, coming on once or twice a week, more often at night, localizing with greatest intensity at the navel. Describes sensation of a rope tied with increasing tightness around her middle. Refers to leg pains as boring and gnawing in character, beginning at the ankles, ascending to the knee and fleshy part of thigh. Sense of tingling and of "things crawling" on the soles of the feet. Of incoordination it may be said that the patient is occasionally inclined to stumble or stub her toe against the floor, especially at times when she feels nervous. Frequently finds it necessary to hurry to the toilet when she least expects it, for fear of

soiling her clothes. Despite the good appetite which she enjoys, she has lost about twenty-five pounds in weight during the past year. (This fact, together with that of a temperature of 100.5° F., led me to suspect a tuberculous focus, which the physical examination revealed as present in the left apex of the lung.) Bowels constipated; sleeps fairly well; says she is more concerned about her eyes than all the other symptoms.

Status Præsens. In general appearance of average height and fair nutrition; bronze-skinned; mouth and pharynx negative for post-specific scars; heart negative; no peripheral arteriosclerosis; pulse regular and full; lungs give evidence of left apical tuberculosis; urine negative; glandular system, isolated enlarged glands, especially inguinal. No bony deformities. Sexual instinct not so keen for past five years—has had "little nature for a man."

Sensations. Subjective: Tingling and formication on plantar surfaces of feet. *Objective*: (1) Tactile sensibility everywhere acute; (2) well-defined analgesia, both legs from knees down; (3) trunk anæsthesia absent; (4) ulnar analgesia slight. No trunk anæsthesia. No persistence of painful impressions. No delayed conduction of sensory impressions.

Sphincters. Slight *incontinentia urinæ.*

Coordination. Virtually unimpaired; no ataxia of gait; no ataxia of station except when put to the test of balancing on one foot. Finer tests, such as heel to knee and finger movements, show slight degree. Tremors absent.

Reflexes. Patellars bilaterally absent; Achilles, bilaterally absent; plantars exaggerated; abdominal, upper and middle zones brisk; Babinski absent; wrist absent.

The Eyes. I am indebted to Dr. Richard S. Patillo for the report of the eye finding, as follows: Total iridoplegia; no oculoplegia. Visual field with "white," one-half inch square; above, two inches; nasal side, two inches; temporal, one inch; lower, three inches; apparently no scotoma. Ocular tension normal. Double optic atrophy. Acuity of vision, O. D. $\frac{20}{100}$; O. S. $\frac{20}{60}$.

Case II.—L. M., aged forty-six years, female, colored, married; color light bronze. The ancestry of this patient is as follows:

Paternal grandfather, full-blooded Indian; paternal grandmother, full-blooded negress; maternal grandfather, full-blooded negro; maternal grandmother, Irish. Thus these strains make this woman one-half negro, one-quarter Indian, one-quarter Irish. Patient born in Ricetown, Maryland, and reared in Baltimore. Parents were free when patient was born.

Of past illnesses, patient records croup and measles when a child, and typhoid fever at sixteen years. Married first at age of nineteen years to a full-blooded negro. Four years married, when she miscarried at two months—no ascertainable cause. First husband died. Married again at age of thirty years, also to a full-blooded negro; second marriage childless. Has no knowledge of a luetic infection. She does, however, state that after the first year of married life, that is, at the age of twenty years, she experienced severe "sick" headaches, coming on with increasing frequency at any time of the day, locating with greatest intensity over the right eye, and again over the left, and accompanied by a sense of nausea, which would occasionally end in vomiting.

Present Illness. Began in 1895, with severe throbbing pains in the legs, neuralgic, as she calls them, and along the nerves, radiating from the thigh downward into the calves. Vision began to fail in 1896. She at that time was under treatment at the Illinois Eye and Ear Infirmary, and was referred by the oculist to Dr. Patrick, who saw her for the first time at the Polyclinic in that same year, and made a diagnosis of tabes. Sight in the left eye was completely lost within six months from the date of onset. At this time incoordination was not very marked. The right eye began to fail her soon after the left, but for the past four years vision has remained stationary. She claims that she can see the daylight out of the corner of the left eye (mental); with the right eye she can distinguish daylight from lamplight or differentiate a bright from a dark day, but she cannot see to read—in other words, she has only "quantitative vision."

Status Præsens. In appearance is fairly well nourished; of bronze complexion; mouth and pharynx normal; thoracic viscera normal; urine negative; sexual instinct indifferent.

Sensations. Subjective: Early in the disease as of "cold water running under the skin," but of late this has disappeared. *Objective:* (1) Tactile sensibility everywhere most acute; (2) analgesia in the limbs, slight from the thigh to the knee, but very marked from the knees downward; (3) trunk anæsthesia absent; (4) ulnar analgesia present.

Reflexes. Patellar: Left abolished, right very faint response with reinforcement; plantar reflexes normal; Babinski absent; wrist taps present; abdominal and skin reflexes present to slight degree.

The Eyes. I am obligated to Dr. Salinger for the eye-findings in this case. Quantitative vision only. Total iridoplegia, both disks gray-white and excavated; double optic atrophy. No ocular paralysis.

Coordination. Gait unimpaired; no static ataxia; slight ataxia to finger movements. Sphincters: Slight *retentio urinæ.*

Case III.—W. F. P., male, black-bronze in complexion, aged forty-nine years, married these ten years—childless marriage. By occupation a waiter. I was unable to ascertain so exact an heredity as in the previous cases. I did determine, however, that the father died an old man from some kidney disease. The mother is living and well, as are two sisters and one brother.

The patient had measles as a child. States that he had gonorrhœa as many as five or six times, the last attack eight years ago. No history of chancre. He has been temperate in his habits, drinking beer moderately, and does not deny taking an occasional whiskey, as he says, to stimulate him since his present nervous trouble has come on. As to injuries, he presents a scar over the left temporoparietal region, which he states is the result of a lacerated wound of the scalp received some twelve years ago from a glass of water thrown at his head.

Present Illness. Onset about four years ago, with dizziness, especially distressing when the patient got out of bed in the morning, and which in a short time reached such an alarming degree as to make his efforts at walking for the first two or three hours of the day almost impossible. In his own words, he could never "gather himself together until about noontime." (I believe that this dizziness is to be regarded more in the light of incoordination than vertigo.) He says that frequently his swaying from side to side was so great as to give onlookers the impression that he had been drinking. During the latter half of the day he

grew steadier on his feet and kept so until the following morning, when the evidences of his incoordination promptly reappeared. A loss of memory has been a conspicuous feature in this patient's case from the very first. Being a waiter by occupation, he found himself greatly handicapped by his poor memory in receiving and remembering orders given him. He would forget parts of an order, and lately he has taken an order as far as the kitchen only to return and ask the customer to repeat same, as he had entirely forgotten it. About one year after the onset of this dizziness and impairment of memory his eyesight began to fail him—three years ago. With this failure of vision there has come a sense of fear when the patient is abroad (born of his ataxia), having fallen time and again over such objects as chairs and other obstacles that seemed to have been in his way. As he puts it, he has "lost his nerve." For the past seven months he cannot see well enough to read print. Fulgurant pains radiating from the thighs into the calves have been present for the past two and one-half years. The patient has regarded these as rheumatic. In addition, he complains of a sense of tightness at the waist, which impression he, of his own accord, chooses to disabuse me of by saying that his trousers are far too large at the waistband to account for this feeling. There can be no doubt that this sense of constriction is none other than a "girdle sensation." Sexual power is weakened to a degree of partial impotence. Has lost appreciably in weight, despite his good appetite. Bowels constipated.

Status Præsens. General appearance that of a man in good nutrition; weight, 145 pounds; height, 5 feet 1 inch. Heart and lungs are normal; no vasomotor disturbance; no peripheral vascular changes.

Sensations. Subjective: Soreness and a sense of burning on the soles of the feet. Says that at times he feels as though he were walking on a gravel path. *Objective* sensations, negative.

Coordination impaired, but only slightly; most manifest when told to turn quickly or suddenly rise from his chair. Not at all present during his more deliberate movements. Romberg sign absent. Slight tremor (coarse) in the right hand upon protrusion.

Reflexes. Left patellar brisk; the right almost abolished. Contrastingly, the left Achilles is abolished and the right Achilles is faintly present with reinforcement. The abdominal skin reflexes are everywhere brisk.

Eyes. (Examination by Dr. R. S. Patillo.) Pupils equal in size, the left slightly irregular in outline. Right pupil reacts to light sluggishly; left pupil does not, but both react to accommodation. Fundus: Optic atrophy in both eyes. Acuity of vision: O. D. $\frac{8}{120}$; O. S. $\frac{0}{00}$; ocular tension normal, no oculoplegia.

Organs of special sense uninvolved. Tongue shows neither deviation nor tremor.

Referring once more to this patient's complaint of dizziness, I do not wish to insist that the patient could not have been suffering from a genuine vertigo in the incipient stage of the disease, because it may be and sometimes is a feature in some cases, but is of distinctly inferior importance and occupies a low place in the scale of conspicuous symptoms. Bonar[11] in 286 cases found it in 0.34 per cent. Another rare symptom presenting in this case is that of tremor, which Bonar[11] found in 1.74 per cent. of his 286 cases.

CASE IV. (in brief).—S. M., female, aged forty years, light brown in complexion (lightest in complexion of all the cases), born of black mother and white father; married and has one living child, prior to the birth of which the patient had two miscarriages, without ascertainable cause. Seamstress by trade. Knows nothing of father; mother living, but patient does not know where. Has one brother and two sisters, all of whom are even lighter in complexion than she is.

Patient had typhoid fever at the age of eighteen years; also vaguely refers to fever and chills. Onset of present illness six years ago, with severe headaches and excessive nervousness. Inquiry as to this nervousness elicited the fact that she had difficulty in threading her needle, although she could see the eye of the needle as plainly as ever before. There were days when she was not annoyed by this symptom, and then for weeks at a time the symptom would reappear. When using the machine her line of stitching was anything but straight at times, and her limbs easily gave out in the act of treading. The symptoms continued until she was forced to give up this kind of work. In the third year of her trouble, eye symptoms developed and for a short period of time prior to the beginning failure she saw things double (diplopia). Bladder disturbance manifested itself about this time. She gives no evidence of disturbance of gait or of severe lancinating pains. She is more worried about her eyesight than anything else, and deplores her inability to resume work as a seamstress. Her symptoms have not taken on an increasingly aggravated form for the past three years. Denies venereal infection.

Status Præsens. A female of slight build, about 110 pounds in weight, and taller than the average woman. Yellow skin; somewhat emaciated in appearance; heart and lungs normal. Urine negative.

Sensations. Subjective: Cramp-like pains in the calves, paroxysmal, but not severe. *Objective:* Analgesia most marked on the dorsal aspect of the feet.

Coordination disturbed and easily detected upon an attempt at finer movements; cannot approximate index fingers well, nor can she grasp small objects without very apparent awkwardness. No ataxia of station except when standing on either foot alone. Gait normal. Hands, more especially the fingers, tremulous upon protrusion.

Reflexes. Both patellars completely absent. Achilles both absent. Abdominal reflexes and skin reflexes faint in their response. Babinski reflex absent.

The Eyes. Pupils equal in size and regular in outline. The irides do not react except to accommodation. Fundus: Partial optic atrophy in both eyes. No ocular paralysis.

Complains of deafness in left ear, which, however, she says is not constant.

I hold it would be erroneous to attach undue importance to the conclusions that accrue from an analysis of so few cases, but that fact should not deter from bringing out and emphasizing points of difference and similarity in the disease as it affects another race than the Caucasian. In the matter of sex the most recent and conservative figures imply a proclivity for this disease in the Caucasian male, and it is a matter of no little interest that in the colored race, respecting my four

isolated cases, the frequency is quite the other way. As to the age at which the disease manifests itself in these cases, it concurs with the statistics that have placed more than 50 per cent. as occurring in the third decade.

The ocular phenomena in this series merit special attention. One of the cardinal symptoms of tabes lies in the condition of the pupils. The Argyll-Robertson pupil was present bilaterally in three of the cases, and in the fourth was found in the left eye, there having been a sluggish reaction to light in the right. The intense pigment in the eye of a colored individual renders a decision as to the presence of a pupillary reaction and its degree of prominence much more difficult than in the eye of a white person. The pupils in all but one case were equal in size and regular in outline. Schaffer,[14] is authority for a remark made by Gowers that the Argyll-Robertson pupil is only present in those cases of tabes which are of undoubted luetic origin. I am not aware that other clinicians coincide with this statement. The failure of vision occurring in all the cases, revealing for its cause the presence of optic atrophy in all of the eyes, led me to look into the question, and I found that relative to the statistics of optic atrophy in tabes as it afflicts the whites there is great variation. Von Grosz[15] found it present in 88 per cent. of his cases; Berger,[16] in 46 per cent.; Dillman,[17] in 42 per cent.; Marina,[18] in 12 per cent.; Bernhardt,[19] in 10.3 per cent.; Thomas, 10 per cent.; Leimbach, in 6.75 per cent. Bonar's figures of 8.74 per cent. are based upon an exhaustive and critical analysis of 286 cases of tabes from the case records of Allen M. Starr's service at the Vanderbilt Clinic, New York. Bonar states that optic atrophy was either well established or beginning in 25 cases, "and," he adds, "one case was that of a colored man in whom the atrophy came on as the first and only symptom except the blindness caused by it and the absence of the knee-jerks." The well-known fact that cases of profound tabes so often come under the care of the oculist for disturbances of vision, and are by him referred for a wider interpretation to the neurologist, led me to investigate ophthalmological statistics for data as to the comparative prevalence of optic atrophy among the whites and blacks.

In the *Archives of Ophthalmology*, 1884, Swan Burnett's[21] treatise on the "Comparative Frequency of Eye Disease in the White and Colored Races of the United States" included a consideration of the diseases of the optic nerve, retina, and glaucoma. His statistics comprehend a consideration of 2325 cases, of which 1514 were colored and 811 were whites, and all of which were by him personally examined and observed. He subdivides his cases into diseases of the conjunctiva, diseases of the cornea, diseases of the iris and choroid, etc., diseases of the optic nerve, retina, etc., as follows:

DISEASES OF THE OPTIC NERVE, RETINA; GLAUCOMA. (SWAN BURNETT.)

	White.	*Colored.*
Atrophy of optic nerve	14	13
Retinitis albuminurica	2	5
Amaurosis	1	7
Retinitis sympathetica	...	1
" specifica	3	3
" pigmentosa	1	2
Neuroretinitis	...	3
Myxoma, optic nerve	1	...
Glaucoma, simple	4	14
" inflammatory	1	2
" secondary	...	1
Hyperæmia retina	2	2
Glioma retina	...	1
Detachment	2	...
	31	54

He fails to comment upon the finding of optic atrophy as a symptom of tabes—in fact, he does not discuss the matter at all. It would be interesting to know what other oculists have to report in this connection.

The cases reported by McConnell[22] in 1899, instances of tabes in the full-blooded negro (according to the author), were of the amaurotic type. In his cases, as well as in mine, all the symptoms, some of which had attained an aggravated degree, were held in abeyance as soon as the optic atrophy had manifested itself. Benedickt[23] goes so far as to say that he had never known an exception to this condition, and Bonar,[24] relative to his twenty-five cases, in which the atrophy dominated the other symptoms, says: "Where the first symptom was the blindness due to optic atrophy there has been no further advance of the disease."

Abeyance of all other symptoms naturally includes an arrest of incoordination, which actually occurs. It is a rare thing for a man with tabes to have marked incoordination and almost total blindness —one or the other may be conspicuous, but seldom, if ever, coexistent. As Dr. H. T. Patrick,[25] in a clinical lecture on "Locomotor Ataxia," says: "This is not a trivial matter, because one patient consulting us for trouble with his legs may know another patient with locomotor ataxia who has become blind, and *vice versa*, and he is greatly worried for fear he may acquire the same symptom. If a patient comes to you with failure of vision from locomotor ataxia, you are justified in assuring that patient that he will always have good use of his legs." Just why this is so has been for years a matter of conjecture. When McConnell presented his five cases before the Neurological Society of Philadelphia, in 1899, emphasizing the abeyance of ataxia in the presence of the optic atrophy, it was suggested in the discussion to invoke the Edinger compensation theory (Ersatz theorie)[26] in explanation of this conciliatory effort on the part of the damaged nervous system. Dr. Spiller, in the discussion, stated that he had entered into an inter-

esting correspondence with Edinger on this point, and could quote a passage from his (Edinger's) letter, which read to the effect that the substitution theory did not supply *all* the needs in the cases to which it applied.

For those less familiar with the Edinger hypothesis, published in 1894, I have given a succinct interpretation of the principles therein involved.*

* Edinger's theory is based upon the time-honored physiological dictum which was original with Weigert, and states that the usage and function of a part implies disintegration and consumption of some of the active tissue. In this sense it has been applied to the nervous system by Edinger, who furthermore assumes that where nutrition in a part is deficient (or even under normal conditions) and the cells are called upon to do excessive work, suprafunction, thus disturbing the balance between combustion and repair, the developmental energy in the resting tissue will serve to bring about a degeneration of the less resistant active parts.

In order that the Weigert-Edinger view as it pertains to both motor and sensory spheres of the nervous system succeed it is essential that it be proved that certain tracts under normal conditions are called upon to perform more work than others, and that these overworked tracts undergo correspondingly pronounced degrees of degeneration. Edinger regards as a prerequisite to the decline a disturbance of general metabolism and a deficient nutrition.

It is observed by Edinger that the sensory nerves, for instance, are constantly transmitting sensations from the skin to the central nervous system; the fact that we are unconscious of the presence of a sensory mechanism does not mean that we are not possessed of one in constant operation, for we are, and the sensory nerves are the very ones, for that reason, to suffer.

From these hypothetical premises are argued the production of the clinical sensory symptoms—anæsthesias, paræsthesias, hyperæsthesias, etc. The sensory disturbances evidence themselves, as might be expected, in those areas on the body where the peripheral nerves are most irritated from contact, for instance, with or pressure of the wearing apparel. Corroborative of this reasoning, there are found in tabes irregular and scattered patches of anæsthesia occurring frequently on the soles of the feet and in a narrow band encircling the waist. This ingenious reasoning Edinger applies to the eyes, more especially to the nerves of the "constrictor iris," since there can be no glance at any object, no matter how momentary, without this muscle and its nerve supply coming into play. The light reflex is almost constantly active, in contrast to which the accomodative reflex is but seldom so.

Reverting once more to the physiological principle, we might expect a degeneration of the fibres controlling the reflex arc to light, while the latter, those governing accommodation, remain normal. In this way the author of the theory chooses to explain the clinical phenomena known as the Argyll-Robertson pupil, optic atrophy, oculoplegias, and especially ptosis. The disturbance in function of bladder and rectum are alluded to in the same sense.

Edinger has drawn a parallel between the above symptoms ascribed to suprafunction in an ill-nourished nervous economy and those which complete the typical picture of tabes, viz., the incoordination, lost knee reflexes, Argyll-Robertson pupil, oculoplegias, optic atrophy, and loss of sphincter control.

If his deductions are correct, the theory supplies a more satisfactory explanation of the pathology of tabes than any as yet offered, and better accounts for the presence of many hitherto less well understood but widely recognized clinical phenomena.

Profound study of the hypothesis thus far constructed led Edinger to carry on experiments in furtherance of the truth of his ideas. He followed in the footsteps of Voss, who produced extreme malnutrition in animals by the use of substances (pyrodin) which impaired the blood-making powers, and then subjected them to forced activities. Animals so treated showed upon post-mortem examination degenerations closely resembling those of tabes in the motor tracts of the cord.

By inference, then, it is clear that Edinger's theory postulates (1) the deleterious effect of noxious agents on the nutritional state of the nervous system; (2) a nervous soil so prepared is further agitated by normal or excessive function until a symptom-complex results, such as we are, for instance, familiar with in tabes.

The theory met with an objection at the hands of Schultze, who argued that if function determined the localization of the disease there would be every reason to note degeneration in the motor nerves and anterior cells, with occurrence of paralysis and muscular atrophy. This objection was overruled by Edinger with the assertion that the motor nerves were ana-

That the Edinger theory as it applies to both motor and sensory spheres of the nervous system succeed it is essential that it be proved that certain tracts under normal conditions are called upon to perform more work than others, and that under the influence of undue stress in the form of overwork, either relative or absolute, these tracts degenerate, this degeneration being associated with a disturbance of general metabolism plus deficient nutrition. In other words, given a man who is drenched with substances of noxious character, toxins, if you will; acknowledge that his entire economy has undergone a metabolic change as a result of their influence, and let him use to excess such faculties or functions as involve certain tracts at the expense of others, and *there* the deficit is most felt, and *there* the degeneration is most evident. People who use their lower extremities most are prone to suffer from ataxia in the legs; people using their eyes most have to contend with optic atrophy. In the course of human events it would be ridiculous to aver that the negro uses his eyes at the expense of his legs or other organs; in fact, it is perfectly right to assume that he uses them far less. Here we encounter a weak point in the Edinger hypothesis. And Edinger himself, true and fair-minded scientist that he is, acknowledges the fallibility in part of his reasoning when he states that his theory may not cover all the needs of a case. There is an experimental groundwork for the substantiation of Edinger's theory, even though the clinical data are not always corroborative. These experiments are highly

tomically safeguarded with a greater power of resistance and a better supply of nourishment than the sensory.

With an acceptance of the Edinger theory much that is vague and indefinite as to the tabes-syphilis question becomes satisfactorily explainable. The normal relation between waste and repair is influenced by the noxious action of syphilis on all the tissues of the body and further impaired by the coincident vascular changes of the disease.

Acknowledging the high percentage of tabetics who are known syphilitics, one is enabled to account for those cases which give neither the history of specific infection nor somatic evidence thereof. By this token we are not called upon to believe that everyone who has tabes must have had syphilis—a fact insisted upon by some authors, but still unproved by reliable statistics.

Edinger has provided a unitary etiological factor for the disease tabes in that syphilis, exposure to cold, wetting, trauma, excesses in "Baccho et venere" are grouped as predisposing noxious features disturbing the nutrition of the nervous apparatus, the associated undue stress of function completing the requirements of the clinical picture.

Even the clinical symptoms bear out the theory: the lower extremities are affected first in a majority of the cases, and in the few cases of tabes high up in the cord, the cervical type or the *tabes cerebrale* of the French, the information was elicited by Edinger that the arms had been called upon to do proportionally more work, and that these patients were by occupation seamstresses, carpenters.

The sex ratio as variously established, from 15 or 8 : 1 in favor of the men, fits well with the theory, since men engage in physical pursuits.

In referring to the pathological process and its limitation to the intramedullary distribution of the sensory neuron and not its peripheral part, which is virtually exposed to the same stress, Edinger explains that (1) the peripheral nerves enjoy the protection of an additional sheath; (2) the least resistant point of the neuron, owing to a constriction of the myelin sheath (or its absence), is at its point of entrance into the cord.

As for a consideration of the therapeutics applied in consonance with this theory, suggestions have been made in the body of this paper.

instructive. By feeding rats pyrodin he caused a profound intoxication and anæmia, after which they were made to indulge in great activities of a certain kind, and it was found that those rats that had been put hard at work showed, post-mortem, a correspondingly advanced grade of spinal-cord degeneration. This discovery led Edinger into the field of therapeutics with a suggestion in prophylaxis, "insisting that exertion and fatigue of any sort should be avoided," maintaining further that, "in a large number of cases treated for a number of years on this basis, results have been obtained in the way of retarding the progressive development of the disease." The proportion of males over females he attributes to this same pernicious effect of overactivity.

I deem it expedient at this point to introduce evidence of very recent date and from a most reliable source, which, to my way of thinking, harmonizes excellently well with Edinger's experimental findings and therapeutic results. A no less distinguished clinician and observer than Marie is responsible for the statement that in his amaurotic cases, in which, *intra vitam*, the ataxia and other symptoms were arrested, post-mortem examination revealed a *less extensive* degeneration in the cord.* Edinger's claim is that bodily overaction increases the degenerative process in the cord and that bodily inaction restricts it. Marie by another route, namely, that of blindness, determines substantially the same fact. In the presence of these data, then, how much nearer are we to a solution of the question, Why does established optic atrophy retard ataxic symptoms? Time was, and only a few years ago, when J. K. Mitchell[27] offered the following explanation: "The blindness does result in a certain concealment of some of the tabetic symptoms—the blind patient, if he can walk at all, necessarily does so without seeing his feet; thus, perforce, he acquires some of the greater ease of motion and improvement in station which the tabetics acquire by learning to execute careful coordinate movements—*not that they have changed the disease tissues of the cord, but* that they use themselves enough better to conceal some of the symptoms of the disorder." Since then Marie's observation has reinforced Edinger's view, and out of the combined issue I believe it is possible to advance a more tenable assertion. The patients with optic atrophy are blind; blind men rather conserve than dissipate muscular energy. They do not overexercise. With them functional wear and tear is at a minimum, and so the disease process in the cord is restricted. A less degenerated cord reciprocally inhibits the other "motor" symptoms. As to why the optic nerve is selected to atrophy in the first place, see note appended on the Edinger compensation theory.

The medical profession has had the Erb-Fournier parasyphilitic postulate so thoroughly impressed upon it that it is unwilling to credit

* A personal communication of Marie to Dr. H. T. Patrick.

any other factor as giving rise to tabes. Some regard, but very little, is shown for von Leyden's conception invoking exposure to wet and cold, and *not* syphilis, as the chief factor in the cause of the disease. Only the fewest are disposed to regard Edinger's theory favorably, although it explains more of the multiform symptoms than any other one hypothesis. Benedickt, who, when he says "*tabicus non fit, sed nascitur*," emphatically denies every factor but that of heredity, meets with little approval at the hands of the profession.

Summing it all up, this much is incontestable, that tabes is a disease of the sensory neuron, caused, in a vast majority of the cases, by syphilis, which, however, does not stand in direct causal relation.

Coming to a consideration of the cardinal cause in the production of the disease in the negro, and conceding an apparently relative immunity in him, who shall deny that Aryan admixture is not accountable for a predisposition in the man, which, plus his vices and acquired venereal infections, has given rise to the appearance of tabes in him? In support of this view I recall that all of the cases heretofore reported, including my own, in which I have been especially solicitous of detailing the ancestry, occurred in hybrids. McConnell, it is true, reports his cases as those of full-blooded negroes, but adopts the view from the premises of color rather than by inquiry into ancestry. A black negro need not necessarily be a full-blooded negro, and I am not willing to allow that mere observation with respect to varying shades of complexion is convincing.

Conspicuous as is the part played by venereal diseases in the life of the negro, syphilis, so very much more prevalent among the colored, is on the increase, and facts supplied from various authentic sources go to prove that the negro has acquired this infection since the period of slavery through an ever-increasing low standard of sexual morality.

While it has proven futile to insist upon a syphilitic etiology for tabes in the Caucasian, nevertheless, I think a causal relationship as it exists for the whites applies in the blacks.

The query as to why there is so little tabes in the negro when there is so much syphilis is a natural and logical one, which may remain unanswered for the same reason that other paradoxes of factual medicine exist and solution of them is suspended. As well might we seek to explain the absence of tabes in Bosnia and Herzegovina, where, as we know, the most intense constitutional syphilis is rampant among the natives. So far as immunity and susceptibility is concerned, I believe that too much has been taken for granted.

As Dr. Matas[8] concludes, "there are no diseases which prevail exclusively in the colored any more than there are diseases which prevail exclusively in the white race."

If any conclusions are to be drawn from a consideration of the various

features touched upon in this paper, they may be summarized as follows:

1. Long residence with the white man has made the American negro anthropologically, physiologically, and pathologically different from his African ancestors.

2. The constitutional variation has been wrought by acclimatization, social environment, and, more than all else, by miscegenation.

3. The influence of miscegenation and the advent of personal liberty are responsible for a new era of diseases.

4. The newer diseases in the negro, of which tabes is an example, are fast becoming more commonly recognized, miscegenation being regarded as the potent factor in reducing the negro's resistance toward disease.

5. Tabes exists in the negro perhaps more commonly than has been supposed, and failure to recognize it may be due to the abeyance or total absence of the ataxic symptoms in the amaurotic type.

6. The Edinger-Marie observations anent the optic atrophy satisfactorily explain that class of cases in which the tabes is arrested by blindnese.

7. Aryan admixture is essential to the production of tabes in the negro.

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REVIEWS.

THE PRACTICAL MEDICINE SERIES OF YEAR BOOKS, comprising ten volumes. By GUSTAVUS P. HEAD, M.D. Vol. IV. Gynecology. Edited by DRS. EMILIUS C. DUDLEY and WILLIAM HEALY. Chicago: Year Book Publishers. March, 1903.

THIS volume is one of a series of ten whose aim is to cover the whole of the year's work in medicine and surgery as represented in current literature. The task of selection—an enormous one—has been most carefully prosecuted, and, while the authors do not assume any responsibility for the opinions expressed, there are frequent criticisms inserted. This critical consideration is the most valuable part of the whole book, as it enables the reader, even if untrained in this particular branch of medicine, to form an intelligent conclusion concerning the subject matter of the article under consideration.

A very complete index adds to the usefulness of the volume, as does also the method of grouping the articles under appropriate headings, as Tumors, Traumatisms, etc. In every case the full reference to the original article is appended, thus enabling the reader to consult the article in the original if he desires. W. R. N.

THE A B C OF PHOTO-MICROGRAPHY. A PRACTICAL HAND-BOOK FOR BEGINNERS. By DR. W. H. WALMSLEY. New York: Tennant & Ward, 1902.

THIS little book should prove of much service to beginners in photo-micrography. The subject is treated in a direct, practical manner; the *little* difficulties, those fatal stumbling-blocks to the beginner in any subject, are pointed out and the way in which they can be overcome clearly shown.

In discussing the microscope and camera, essentials in the "making of a *ma*croscopic photograph of a *mi*croscopic object," the author dwells not only on the manner of doing work with the best apparatus, but also —and this is so important for the majority of students—on the good work that can be done with the very modest appliances possessed by many (medical students and others). The question of what illuminant to use is gone over pretty thoroughly, and, while the preference is given to acetylene, we notice that the author has a very kindly feeling for the easily obtainable coal-oil lamp. Perhaps the best chapter is that devoted to "Negative Making," and in this the author, in a cheery, earnest way, fairly carries the student along with him. The entire pro-

cess of making negatives, working with high and also with low powers, is gone over step by step until the negatives are left drying in the rack. Some of the plates shown are excellent.

Another chapter, devoted to "Printing Methods; Lantern Slides," does not call for particular comment except that here, as elsewhere, the author has again made an earnest and, we think, successful effort to point out many of the difficulties incident to the work and to indicate the most practical means of overcoming them. H. M. W.

CANCER AND TUMORS OF THE STOMACH. By SAMUEL FENWICK, M.D., F.R.C.P., and W. SOLTAU FENWICK, M.D. Lond., M.R.C.P. Philadelphia: P. Blakiston's Son & Co., 1903.

THIS book is the result of a painstaking, statistical, and clinical investigation of the subject of carcinoma of the stomach. It makes no pretensions to any original views regarding the pathology or etiology of cancer, but presents merely the current teachings upon these subjects, with some very good illustrations of rare specimens. To our mind it seems rather unfortunate that the authors should have adopted the method of percentages for recording their results. It is of little value, after all, to know that in 48 per cent. of their cases the pain was severe or continuous; that in 38 per cent. it was occasional; that vomiting occurred in 87 per cent. of the cases; that hemorrhage was present in 34 per cent. and pronounced anorexia in 32 per cent. Such statements are of too little value when taken by themselves. It is really the grouping of symptoms rather than their comparative frequency that enables us to make a diagnosis or to form a prognosis. This appears to be the most serious criticism that can be made of the book. It is full of careful clinical observations, of accounts of cases intelligently studied; it takes an exceedingly rational view of the subject, and the statements impress the reader as being trustworthy. Among some of the interesting details of the work may be mentioned that the authors express a tacit approval of Hemmeter's method of curetting the wall of the stomach with a sharp-edged stomach-tube in order to obtain fragments of the mucosa for microscopic examination. They lay great stress upon the presence of irregular mitoses in the cells in the diagnosis of cancer. They do not regard the presence of enlarged glands above the clavicle as of much value in the diagnosis, and this accords entirely with our own experience. On the other hand, evidence of extensive metastasis to the superficial lymph glands is, of course, always of great significance The list of clinical varieties is perhaps more confused than is entirely satisfactory, and yet it is difficult to see how this could be avoided unless several forms of classification were adopted. It may be interesting to mention the different varieties: 1, carcinoma of the cardia; 2, carcinoma of the body; 3, carcinoma of the pylorus; 4, total infiltration; 5, the latest form; 6, the ascitic form; 7, the dyspeptic form; 8, the anæmic form, of which three subvarieties are recognized; 9, carcinoma originating in simple ulcer; 10, carcinoma of the stomach in early life. The subject of the differential diagnosis is carefully studied, particularly with reference to the different clinical forms. The chapter on Treatment is, however, one

of the briefest of the book, and is only an added illustration of our practical helplessness in the management of this disease. Lavage, rectal feeding, diet, and a few drugs for the relief of the symptoms are suggested, and then the only thing left is surgical intervention. The discussion of the indications and the contraindications for this might have been more complete; but as it is, it is fairly satisfactory. The second part of the book deals with sarcoma and other non-carcinomatous tumors of the stomach, including foreign bodies, such as hair balls, gastroliths, etc., and carcinoma and other tumors of the duodenum. To each chapter there is appended a bibliography, and there is also a very extensive bibliography upon the subject of cancer of the stomach. This greatly enhances the value of the book. It is beautifully bound, printed, and illustrated, and the index is complete without being too large. Taking it altogether, it is a valuable contribution to the subject of gastric disease. J. S.

NOTHNAGEL'S ENCYCLOPEDIA OF PRACTICAL MEDICINE. American edition. "Diseases of the Liver, Pancreas, and Suprarenal Capsules," by Professors Oser, Neusser, Quincke, and Hoppe-Seyler, with additions by Professor Reginald H. Fitz and Frederick A. Packard, M.D. "Diseases of the Stomach," by Franz Riegel, M.D., edited with additions by Charles Stockton, M.D. Authorized translation from the German, under the editorial supervision of ALFRED STENGEL, M.D. Philadelphia, New York, and London: W. B. Saunders & Co., 1903.

THIS great work has long been a standard medical encyclopedia in the professional world, but as its precious contents have been accessible only to those who are familiar with the German tongue, the copies of the work on this side of the Atlantic were so scarce as to be practically found only in the larger medical centres of the country. Its translation, therefore, and publication in the form of an American edition has been the source of great gratification to the American profession. In choosing the editors who should have charge of the individual volumes as they appeared in this country the very best men in the various lines have been procured. These American editors have not contented themselves with simply supervising the translation of the volumes from the German, but have incorporated into each book the results of the work of their English and American colleagues. The effect has been to transform a purely Teutonic into an international work. In the first of the volumes under consideration this improvement on the original work is rendered particularly noticeable by the incorporation of the results of the work of Opie and Flexner within its pages. Although German investigators had long ago begun the study of pathological conditions of the pancreas, their relative frequency and importance has only been realized within recent years, and there is no doubt that the lecture by Dr. Reginald Fitz, one of the editors of this volume, upon "Acute Pancreatitis," which was delivered in February, 1889, was responsible for much of this recently aroused interest in the subject. The fact that he is the most eminent clinical authority on pancreatic diseases in this country lends a peculiar value to his services in the present instance. The section on Diseases of the Liver was the last literary work of its

lamented editor. It exhibits the same care and learning which was the peculiar property of all the literary work of Dr. Packard, and serves to emhpasize the loss which the American medical profession has sustained by his premature death.

Professor Riegel's work has been most ably edited in the American edition by Dr. Charles G. Stockton, of Buffalo. Although the work throughout is most admirable, yet for special commendation we would mention the part which deals with the analysis of the stomach-contents. This subject is now so all-important in the diagnosis of pathological conditions of that organ that it is of great value to have so authoritative a statement of the various means that are employed. In a work so complete as the present, one cannot but regret that somewhat more space has not been given to the surgical treatment of gastric diseases. This is the one criticism which we would offer of an otherwise most excellent book. The present volumes do not contain quite the wealth of illustrations which was so noticeable in those previously issued. They are, however, accompanied by plates which amply illustrate the topics under discussion. Both volumes contain large numbers of tables, and are particularly noticeable for the complete bibliographies appended to their various sections. J. H. G.

EXPERIMENTS ON ANIMALS. By STEPHEN PAGET, with an Introduction by LORD LISTER. New and revised edition. New York: G. P. Putnam's Sons. London: John Murray, 1903.

WE are all familiar with the saying, "Oh that mine enemy would write a book!" and there is no doubt that a large amount of the material which is put forth in controversial literature acts as a boomerang to its authors. Although we trust we have enough intelligence to be entirely out of sympathy with the fanatical vagaries of the antivivisectionists, it was with almost a feeling of regret that we picked up this book, knowing that it would be regarded as a contribution to this aspect of the warfare of science with ignorance. Its perusal, however, has instilled us with profound gratitude to Paget for the great service which he has performed in its compilation. He has not attempted to answer any of the so-called arguments of the antivivisectionists, but has contented himself with a plain, historical *résumé* of experimentation upon animals in different lines of scientific work.

The first section of the book deals with the history of the experiments by which physiology was raised to the rank of a science and the various functions of the body studied and ascertained by the labors of countless experimenters upon lower animals. The second section considers experiments in the realms of pathology, materia medica, and therapeutics. All the wonderful advances made in our knowledge of tuberculosis, diphtheria, tetanus, typhoid fever, of the properties of snake venom, and in the proper understanding of the transmission of malaria, yellow fever, and various parasitic diseases, are detailed in a graphic and scientific manner, so that the mere recital proves the absolute necessity of animal experimentation to the advancement of medical knowledge.

The last part of the book is devoted to detailing the operations of the law relating to experiments on animals in Great Britain and Ireland.

It is a pity that a work of this character cannot be more widely circulated among the laity. It would hardly be worth while to attempt to popularize it among the antivivisectionists, as that purblind section of the community is hardly worth the effort at conversion, and is only aided in its mischievous work by any attention bestowed upon it. If, however, the ordinary layman could be brought to read, learn, and inwardly digest its contents he would be much less likely to be disturbed by the clamor of the ignorant, who assume that there is another side to this one-sided question. F. R. P.

A MANUAL OF DISEASES OF THE EYE FOR STUDENTS AND GENERAL PRACTITIONERS. By CLARENCE A. VEASEY, A.M., M.D., Demonstrator of Ophthalmology in the Jefferson Medical College, Assistant Ophthalmic Surgeon to the Jefferson Medical College Hospital, Ophthalmic Surgeon to the Methodist Episcopal Hospital, and Consulting Ophthalmologist to the Philadelphia Lying-in Charity. Illustrated with 194 engravings and 10 colored plates. Philadelphia and New York: Lea Brothers & Co., 1903.

IN this book of 412 pages (19 chapters) Dr. Veasey gives a lucid exposition of the present state of ophthalmology as fully as this can be done in these limits. As the author truly states, the difficulty consists in deciding what is best to include, the amount of material being so great. The choice has been very judiciously made. The writer has succeeded in the purpose he set for himself, namely, "to present in a systematic, practical, and concise manner those facts concerning diseases of the eye which will be of most service to students and practitioners of medicine."

A limited treatise upon a subject of the extent of modern ophthalmology can treat only of the most salient points. If these are judiciously selected, clearly stated, and the views in general sound, everything has been accomplished that can be expected of such a work. The book before us entirely fulfils these conditions. It deserves and will, no doubt, rank with the best works of its class. The reader who has mastered its contents will have to go to the larger and more pretentious works to extend his knowledge of ophthalmology. He will have gained from Dr. Veasey's book all that is possible from a work of its scope.

T. B. S.

ESSAYS ON CLINICAL MEDICINE: being reprints of papers published at various times in THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES by BEVERLEY ROBINSON, A.M., M.D., Paris. Philadelphia: William J. Dornan, 1903.

INTO this volume Dr. Robinson has gathered eleven articles on practical medicine, which he has contributed to the columns of THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES during the last fourteen years. Seven of these essays deal with the diagnosis and

treatment of various cardiac disorders, and it is a great pity that they cannot be in the hands not only of all practitioners, but of every student of medicine. Their broad and philosophic scope, their very practical nature, and the ease with which they are written render them not only instructive and profitable to the reader, but really delightful reading as well. There are few clinical teachers who are better qualified than Dr. Robinson, and the ease and charm with which he writes has an individuality all its own.

Of the rest of the essays the first article in the book is on "Creosote as a Remedy in Phthisis Pulmonalis," and is a model of the way in which the true therapeutic value of any remedy should be studied and written of.

In the essay on the course of the "Treatment of Certain Uræmic Symptoms" Dr. Robinson deals with some of the various types under which uræmia may appear, and with the suitable treatment to be followed under different circumstances.

The article on the "Etiology and Treatment of Certain Kinds of Cough" considers some of the more obscure causes of their symptoms, and is most instructive.

The last two articles, dealing with tuberculosis, pericarditis, and cirrhosis of the liver, are fully up to the standard of the others in interest and charm.

Dr. Robinson is to be heartily congratulated upon the collection of articles which he has gathered together in this work for the benefit of the medical profession. F. R. P.

A Reference Hand-book of the Medical Sciences: Embracing the Entire Range of Scientific and Practical Medicine and Allied Science, by various writers. A new edition, completely revised and rewritten. Edited by Albert H. Buck, M.D., New York City. Volume VI. Illustrated by chromolithographs and 763 half-tone and wood engravings. New York: William Wood & Co.

The revised edition of the *Reference Hand-book* is now almost all published, and it is gratifying to note that this new issue is not merely a reprint of the previous edition with a few additions, but a complete revision of the whole work. All the articles bear evidence not only of new work on the part of their various authors, but also of careful editorial revision. There is no work in the English language which in any way equals this publication in its encyclopedic value to the physician. It is, in reality, a series of well-written monographs on every conceivable topic in medical science. It is impossible in a volume which contains so many articles—most of them of so much importance—to single out for particular mention any individual one. Among the groups of articles which will prove of value in special lines of work we would especially notice those on Ophthalmology, on the Nose and Throat, and on the Nervous System. The alphabetical arrangement of the work is such that most of the articles bearing upon these topics are included in this volume of the series.

One particularly noteworthy feature of the plan of the *Reference Hand-book* is the excellent short bibliographies which accompany the

more important articles. These bibliographical notes are so short that they do not interfere with the space which should be given more particularly to the body of the article, but the references in them are so well chosen that, as a rule, they form a most excellent and reliable guide to the leading publications on the subject under discussion. This makes the work of the greatest service to contributors to medical literature.

Wood's *Reference Hand-book* is not only a work of which its publishers should be proud, but is to be regarded as one of the most valuable outcomes of scientific medicine in America. The names of its contributors include many leading workers in the various departments of medicine and surgery in America. The book is one which should be in the library of every medical man who attempts to keep up with the progress of his profession. F. R. P.

EYE SYMPTOMS AS AIDS IN DIAGNOSIS. By EDWARD MAGENNIS, M.D., D.P.H., late Clinical Assistant at the Royal London Ophthalmic Hospital; author of *The Eyesight of School Children*, *Hygiene in the School Room*, *Healthy House Sites*, and *The Irish Poor Law System*, etc. Bristol: John Wright & Co. London: Simpkin, Marshall, Hamilton, Kent & Co. Limited, 1903.

IF brevity be the soul of wit, this diminutive booklet must be given a high place. It contains 74 pages of text, 3¼ by 2¼ inches per page of print, plus 11 pages of an ophthalmic glossary. It is far too brief to be of use to the ordinary practitioner; at the same time it has a value for the physician who has paid some attention to ophthalmology, for it is a well-written and terse statement of some important ophthalmic conditions relating to general diseases. The reactions of the pupil in health and disease, palsies of the ocular muscles, fundus lesions, and the different varieties of hemianopsia as focal symptoms of cerebral lesions—all these important matters are quite fairly considered. T. B. S.

DISEASES OF THE SKIN. A MANUAL FOR STUDENTS AND PRACTITIONERS. By ALFRED SCHALEK, M.D., Instructor of Dermatology, Genito-urinary and Venereal Diseases, Rush Medical College (in affiliation with the University of Chicago), Chicago, Illinois.

THIS little book of about two hundred pages is an accurate, but extremely brief, presentation of the more important facts of dermatology. The various diseases of the skin are considered in alphabetical order, their symptoms, pathology, causation, and treatment being briefly outlined. To facilitate the use of the book as a quiz compend a series of questions upon the more important points are arranged at the end of each section. While of little use to the practitioner, since it is nothing more than the briefest outline of the subject, the manual will probably be found useful by the student if used in conjunction with one of the larger treatises. M. B. H.

THE ELEMENTS OF PATHOLOGICAL ANATOMY AND HISTOLOGY FOR STUDENTS. By WALTER SYDNEY LAZARUS-BARLOW, B.A., B.C., M.D. Camb., F. R.C.P. Lond., Pathologist and Lecturer on Pathology at the Westminster Hospital. Author of *A Manual of General Pathology*. Philadelphia: P. Blakiston's Son & Co., 1903.

THE present volume, designed as it is for the use of students, is an excellent example of the best type of this class of text-book. The general treatment of the subject is fundamentally good. The descriptions of various pathological processes, though short, are concise, and in almost every instance the important points are clearly brought out, while the confusing details are suppressed. Many of the illustrations lend personal tone to the book, a feature which is so often lacking in similar works, and, in spite of the fact that some of the cuts are not quite up to the standard, none are stereotyped or hackneyed. Bacteriology is very wisely omitted. The pathology of certain organs, for instance the spleen and lymph glands, is, perhaps, too briefly treated, and occasionally, as in the case of typhoid fever, the histological changes are not described in such detail as one might wish, and yet little that is important is omitted. Theoretical discussions are carefully avoided, and no conflicting authorities are quoted. Indeed, the total lack of reference to any literature upon pathology does not impress one as being altogether stimulating for the student. The book is divided into two parts. The first deals with general pathological anatomy and histology, while the second is confined to a discussion of the pathological anatomy and histology of special organs and tissues. In the first part the sections on Inflammation and New-growths are especially noteworthy. In the second part the pathology of the various organs is discussed in turn. The section on Bones is particularly good. The addition of such special subjects as the skin, eye, and ear is very useful. The pathology of the nervous system is shortly and concisely treated. Certainly the intricate problems concerning nerve degeneration belong strictly to the domain of the neuropathologist, and the author has not dealt to any extent with these conditions. The section on the Nervous System, therefore, keeps its due proportion in the work as a whole. Altogether the book is valuable and forms a most excellent introduction to the study of pathology. It is well written, well printed, and supplied with a full index. W. T. L.

THE PRACTICAL MEDICINE SERIES OF YEAR BOOKS, comprising ten volumes on the Year's Progress in Medicine and Surgery. Issued monthly. Under the general editorial charge of GUSTAVUS P. HEAD, M.D. Volume III. The Eye, Ear, Nose, and Throat. December, 1902. Chicago: The Year Book Publishers.

IT is a pleasure to add to the tribute of praise which has been extended to this excellent little series of year books. The present volume is fully up to the standard of its companions, and maintains the place so well won by previous issues. It contains rather more illustrations than in other volumes of the series, which add very materially to its value. F. R. P.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Trypanosomiasis in Man.—Until within the last year it was believed that diseases due to infection with some form of trypanosoma were confined to the lower animals. Up to the present time four well-known diseases occurring in lower animals have been shown to be associated with the presence of trypanosomes in the blood. These are:

1. Surra, the organism which was discovered by Dr. G. Evans, in 1880, in horses and other animals in the northwest provinces of India.

2. Nagana, a disease attacking horses, cattle, and other animals in Central Africa and other regions. This form of the parasite was discovered by Bruce in 1894. The disease is transmitted by the tsetse-fly, and is also known as the tsetse-fly disease.

3. Mal de caderas, very similar to surra and nagana, occurring in Central America and Brazil, and produced by a trypanosoma probably identical with T. brucei.

4. Dourine, or maladie du coït, occurring in Algeria, Southern France, Spain, and Turkey, the pathological agent of which is the T. equiperdum (Doflein), T. rougeti (Laveran).

There has been considerable controversy as to who deserves the credit of having first detected the presence of trypanosomes in the blood of human beings. It is only just to say that Nepveu, while studying malaria in Algeria between 1890 and 1898, discovered a flagellate body in the blood of a human being which he recognized as being a trypanosome. His observations were not published until 1898 (*Comptes Rendus des Séances de la Société de Biologie*, December 24, 1898), and it does not seem to have attracted much attention. The attention of those interested in tropical diseases was not specially directed

to human trypanosomiasis until the report of a case by Dutton (*Thompson Yates Laboratory Reports*, vol. iv., part ii., May, 1902, and *British Medical Journal*, September 20, 1902, p. 881) and Forde (*Journal of Tropical Medicine*, September 1, 1902). This case occurred at Bathurst, Gambia, and had been for some time under Forde's observation, and was referred to Dutton, who had been sent out by the Liverpool School of Tropical Medicine to study the source and distribution of mosquitoes in the town. The patient was not a native, but an Englishman, in the employment of the government.

Trypanosomiasis, as illustrated by this and subsequently described cases, is characterized by well-marked general wasting and weakness; œdema of the face, trunk, and legs; patches of multiform erythema of the trunk and extremities; frequent pulse and respirations; marked enlargement of the spleen; moderate anæmia; and irregular fever of a relapsing type. The disease is extremely chronic in character.

In fresh blood the parasite appears as a very minute, worm-like organism, difficult to detect with a magnification of 300 diameters. It glides fairly rapidly among the red cells, imparting very little motion to them. One end is drawn out into a whip-like process—the flagellum. The other end is bluntly conical. Attached along one side of the body is a transparent, flange-like process—the undulating membrane. A refractile spot (vacuole) is situated toward the posterior end. The parasite when in active motion moves in a screw-like manner, its body rotating around the longitudinal axis. The organism stains by a modification of the Romanwsky method, and has a nucleus situated a little anterior to the middle. A micronucleus or centrosome is also demonstrable in the neighborhood of the vacuole. In stained specimens the parasite averages 22μ in length and 2μ to 2.8μ in breadth.

Dutton made no inoculations of the human blood containing these trypanosomes into animals, and was not able to prove its identity with the trypanosomes occurring in lower animals. Specimens were examined by Laveran, who did not think that it was identical with any of those seen in lower animals. Dutton, therefore, thinks that it is a new species, and suggests the name trypanosoma gambiense. He believes that it is unquestionably the cause of the symptoms manifested by the patient.

Manson and Daniels (*British Medical Journal*, May 30, 1903, p. 1249) report an almost identical case in a woman missionary, aged forty years, who had been referred to them after she had returned from the Congo Free State. An interesting point in the history of this case is that she was bitten in the leg by some insect two weeks previous to the onset of the symptoms. The differential count of the leucocytes in this case showed a great increase of the large mononuclears at the expense of the polymorphonuclears and eosinophiles. The parasites were uninfluenced by quinine, arsenic, and methylene blue. Having found that horses were not susceptible to inoculations of this trypanosome, the writers attempted treatment of the case by injecting horse's blood serum into the subcutaneous tissue, without any effect on the organisms and with rather disastrous effects on the patient's general condition.

In support of the view that trypanosomiasis is of rather wide distribution in Africa is the fact that Baker (*British Medical Journal*, May 30, 1903, p. 1254) has recently reported three cases, all in natives, from Uganda. These, however, were mild and of short duration.

Castellani (*The Lancet*, June 20, 1903, p. 1735) has made a very important observation regarding the etiology of "sleeping-sickness" in Africa. His observations were made in Uganda. In thirty-four cases of sleeping-sickness trypanosomes were found in the cerebrospinal fluid obtained by lumbar puncture in twenty cases. The species was very similar to if not identical with the T. gambiense of Dutton. After Castellani left Uganda his investigations were continued by Bruce. The results in his series were even more remarkable. The trypanosomes were found in the cerebrospinal fluid in every one of thirty-eight cases of sleeping-sickness, and in the blood in twelve out of thirteen cases of the same disease.

Trypanosomiasis occurs in rats in this country, and Novy, of Ann Arbor, reported at the recent meeting of the American Medical Association that he had grown the parasite in pure culture on rabbit's blood serum mixed with agar. The growth does not occur on the surface of the nutritive medium, but in the water of condensation.

On the Etiology of Variola.—The organism which COUNCILMAN, MAGRATH, and BRINCKERHOFF (*The Journal of Medical Research*, May, 1903, p. 372) believe to be the cause of smallpox apparently belongs to the sporozoa class of the protozoa. It appears first in the lower layers of the epithelial cells of the skin (before there is any anatomical evidence of vesiculation) as a structureless body from one to four microns in diameter. These bodies, one or more in number, lie in vacuoles in the cells. They gradually increase in size, and granules, embedded in a reticulum, appear within them. A definite nucleus has not yet been seen. The body now segments into a number of round bodies one micron in diameter. At the time of segmentation, and when most of the intracellular bodies have disappeared, small round or oval, ring-like bodies appear in the nucleus. These increase in size and acquire a definite structure, consisting of a series of vacuoles around a large central vacuole. The intranuclear body eventually entirely destroys the nucleus, and later the cell itself ruptures, setting it free. The intranuclear body becomes transformed into a number of circular, ring-like bodies with a central dot. These are evidently spores from segmentation of the intranuclear body. The investigators regard this intranuclear body as a further stage of development of the intracellular body, and as representing a second complete cycle of development. It is the spores of the intranuclear body which they regard as the infecting material of variola. In vaccination of the rabbit and calf Dr. Tyzzer has found only the intracellular forms. The inoculation of the rabbit with the contents of the variola pustules gives rise to bodies similar to those in vaccinia and in the first cycle of variola. Here also only the intracellular bodies have been found. Inoculation of the monkey with contents of variola pustules produces lesions which they regard as variola, and in these both the intracellular and intranuclear cycles are found, occurring in the same sequence as in man. From these experiments they regard it as extremely probable that in smallpox the complete development of the parasite through two cycles takes place, and that in vaccinia the primary cycle only. In the contents of the vesicles and pustules they have seen forms of the intranuclear bodies which seem to show that the intranuclear cycle is sexual in character.

SURGERY.

UNDER THE CHARGE OF

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The Treatment of Prostatic Hypertrophy.—Rovsing (*Archiv f. klin. Chir.*, Band lxviii., Heft 4), in an admirable and exhaustive consideration of this important subject, states that the first method of treatment was by catheterization, which not only had its disadvantages, but by reason of its danger soon fell into disuse. Then came the method of Bier, based on the false presumption that the prostate of man is analogous to the uterus of woman, and that the hypertrophy of the former is a myomatous condition. The danger of this method was clearly pointed out when the statistics showed a mortality of 30 per cent. Then came Ramin and White, who proposed and recommended castration. This method was based on sound theory as well as on the results of animal experimentation, which latter showed that the removal of one testicle was followed by a decrease in the size of the corresponding half of the prostate. But this method was not constant in its results; some cases were benefited and still others unimproved, and about 50 per cent. were cured. Then Isnardi, in 1895, recommended vasectomy. The principle is the same as that of castration, only, the testicles not being removed, the cosmetic effect is better. The operation has also proven itself to be less dangerous, as it may be performed without a general anæsthetic. Trendelenburg, in 1897, revived the same operation as Bottini had proposed in 1874, and which bears his name—the galvanocautery incision. So if one refers alone to the literature the proper treatment of any given case becomes a great question, but it is to be remembered that each method has its value, and the remaining question to be settled is, under what circumstances and which surroundings, which operation is the best; for no one method is the best treatment for every case. The view of Cinkanowski, that a kind of antagonism exists between the gonorrhœal inflammation and prostatic hypertrophy, is not borne out by a careful consideration of the history of many cases and by histological examination. The greater majority of prostatics belong to the minority of individuals who have not had gonorrhœa. Prostatic hypertrophy is only a senile condition, and it is a proof of the weakening of the sexual apparatus. This seems like a contradiction to say that a hyperplasia of the glandular tissue of this organ is the result of senile degeneration when this, as a rule, is shown by atrophy in other organs. What is the function of the prostate under normal conditions? It is, as Türbringer has clearly shown, the secretion of a fluid which is a necessary adjunct to

the motility of the spermatozoa. That hypertrophy does not take place in order to make up, as it were, for a decrease in the semen is clearly shown by the fact that an atrophy of the gland takes place when castration or vasectomy is performed.

The author reports a series of 126 cases treated by catheterization, in all of which there was a medium grade of hypertrophy. In the first group are 7 cases in which there was practically total retention with pain, and in whom an immediate operation was not advisable. In the second group are 10 cases which were characterized by partial retention, with frequent urination and pain. These were catheterized at intervals, and in two to three weeks had progressed so that they could wholly empty the bladder, but in a few more weeks or months the old symptoms of pain and partial retention returned. In the next group of 91 cases the symptom of retention was not benefited and the treatment was only palliative. In the next group of 15 cases, in which partial retention was the greatest symptom, the catheterization did harm and the patients were unable to spontaneously evacuate the bladder. In the 116 of the 126 cases the catheter treatment had absolutely no beneficial effect on the retention, and in these patients came the question whether or not to begin catheter life or to operate. In 11 cases the urinary passages were so infected by the catheterization that operation was out of the question, and all of them eventually died as the result of an infective pyelonephritis. The statement that prostatic hypertrophy can be cured by catheterization is thoroughly unwarranted.

Five cases of double-sided castration are next reported. Briefly stated, the first patient was aged eighty-five years, and had not had gonorrhœa. For the past eleven years he had catheterized himself four times daily, as he had complete retention. An examination showed that the prostate was enormous, though soft. Five weeks after the operation he passed his urine voluntarily, and continues to do so. The second case, a man of seventy-six years, with a negative history as regards gonorrhœa; catheter treatment of no benefit; suffering from a medium-sized hypertrophy, with a residual urine of 200 to 300 cubic centimetres. The patient made a complete recovery, and when seen six years later seemed to be perfectly well. The third case, a man of sixty-four years, with a negative history as regards gonorrhœa; catheter treatment was of no benefit. History of partial retention and painful urination, with about 200 cubic centimetres of residual urine and a large, sanguineous prostate; made a complete recovery, and when seen six years later was apparently perfectly well. The fourth case, a man of fifty-six years, had a previous history of posterior urethritis and prostatitis. He was worse after catheterization, and had 970 cubic centimetres of residual urine, and his condition was such that it was not advisable to fully empty the bladder. There was complete retention, and the prostate was densely hard, fibrous, and somewhat enlarged. When seen three months later his condition was unchanged and he voided all his urine through a catheter. The last case, aged seventy-eight years, with a previous history of gonorrhœa, had had an ammoniacal cystitis secondary to catheterization. The prostate was hard and of a medium degree of enlargement, with 200 to 300 cubic centimetres of residual urine. The operation was without benefit, and the patient is obliged to use a catheter regularly. As one can see, the result of these

few cases of castration is in no way bad—60 per cent. were cured. A further analysis of these cases shows that the operation in all the cases had a beneficial effect on that most distressing symptom—retention. In the two patients in whom the operation was apparently without influence in decreasing the size of the prostate an inquiry into their previous history shows that each had had gonorrhœa, with a resulting posterior urethritis and prostatitis. In these cases the prostate was hard in consistence, fibrous in texture, and tightly adherent, while in the other cases which had no venereal history the prostate was soft and sanguineous or boggy and freely movable.

The results of 40 cases of double retention of the vas deferens were 27 cured, 9 improved, and in 4 cases no appreciable changes. By the term cured is not meant a disappearance of the hypertrophy, but instead a disappearance of such symptoms as pain, with the reduction of the residual urine to a minimum quantity. Three of these cases after an interval of six months, one and two years, respectively, have had a recurrence of their symptoms, and have required a Bottini operation for their relief. The other 24 cases have remained well during the following periods since the operation: 3 cases, six years; 4 cases, five years; 4 cases, four years; 1 case, three years; 5 cases, two years; 2 cases, one year; and 5 cases, from six to nine months. So one sees that in 60 per cent. of these cases the patients were relieved of their disagreeable symptoms for a ranging period of time, but one cannot say that this result is permanent, for in one case there was a return of the symptoms after an interval of two years. Still one may hope that the results from this operation will be permanent, for in a greater proportion of the cases the patients are still well after an interval of from four to six years. In none of these cases were there any serious sequelæ after the operation, and the only death soon after the operation was that of a man, seventy-five years old, who suffered from a severe nephritis, and who died of uræmia fourteen days later. In the mean time, as the result of the operation, the severe hemorrhages from the prostate, which had been his most troublesome symptom, had ceased. In addition, this operation avoids the mental anguish which so often follows castration. In many of the cases the prostatic hemorrhage stopped almost immediately after the operation. The retention was also improved, but in two to six months it again became a noticeable symptom. In two cases a Bottini operation was done later, and in two others cystostomy, and in five others the improvement, though marked, was not constant, and they were obliged to urinate often and with difficulty. In 10 per cent. of the cases the operation was without result, and these four patients later came to cystostomy. These cases were all characterized by a tremendously large prostate, with a swollen bladder and total retention.

Fourteen cases were subjected to the Bottini operation, and of these 2 were cured, 5 improved, 6 made worse, and 1 died; or, in other words, only 14 per cent. were cured, not quite 36 per cent. were improved, 43 per cent. were made worse, and 7 per cent. died. More careful consideration of this list shows that in what at first sight seems to be two cured cases the residual urine was 200 and 150 cubic centimetres, and in the first of these cases the post-operative period was characterized by the dangerous symptoms of chills and high fever, as the result of an infection of the prostatic wound from an old cystitis. In the second case a vasectomy had been per-

formed eleven months before, and for six months afterward the patient had improved, but then began to be worse again, and the Bottini operation was followed by a most satisfactory result. There were two cases of hemorrhage in the second group, due to a failure of technique in having the knife at white heat instead of red hot. It follows as a matter of course that burns are at best a hard wound to heal, and this is especially true in the case of the prostate, where the wound is constantly in contact with infected urine and also at a point where there is a sphincter. The cases that are not followed by pain and irritation on urinating after this operation are those where the operation has been of very little value, as the median lobe of the prostate has not been involved in the hypertrophic process, and the wound made by the knife soon heals.

Partial suprapubic prostatectomy: The author reports 6 cases in which he made a suprapubic extirpation of the median lobe with most satisfactory results.

Cystostomy: There were 21 cases, of which none died from the operation, but 4 died shortly afterward of advanced kidney disease, from which they suffered, while the others still enjoy good health. In 3 of them urination was normal at the end of six months. In 1 case vasectomy was performed simultaneously with the cystostomy, and in 2 others a Bottini operation had been performed a few weeks before. The author believes cystostomy to be indicated when a large, swollen bladder is present, whose contractile power is either very slight or absolutely wanting; when catheter life is so hard that it becomes a burden; in the presence of an incurable cystitis, and in the presence of repeated attacks of hemorrhage from the prostate.

Conclusions. The author states that if called upon to treat a patient who has never before been under treatment, and who suffers from a mild degree of partial retention, or perhaps total retention, the first thing to try is regular catheterization. In a large proportion of cases this is followed by very great relief to the patient, but one should not forget, and should also make the patient clearly understand, that this method of treatment will not result in a cure. If, however, the case is one with a markedly swollen bladder, or, in other words, there is a large amount of residual urine, the best treatment is a vasectomy as soon as possible, and eventually prostatectomy, for in these cases catheter treatment is usually of no avail, and is often dangerous from the introduction of micro-organisms. In those cases where the patient has used a catheter for a long while and suffers from either partial or total retention, a vasectomy, followed, if necessary, by prostatectomy, is the operation of choice. In those cases where there is total retention with cystitis, which, on catheterization, have hemorrhage, chills, and fever, a cystostomy with a simultaneous or subsequent vasectomy is to be recommended. As a result of the vasectomy the prostate will atrophy, and then an attempt should be made to close the fistula. If normal urination is not possible as the result of vasectomy or castration, then one has the choice between cystostomy and regular catheterization. The latter is to be recommended in those cases who, by reason of education and environment, are enabled to carry out the treatment carefully, and who do not find catheter life too trying. On the other hand, when the patient belongs to the poorer classes, cystostomy is to be recommended. It is to be remembered that in the use of the catheter

there is always the danger of infection and hemorrhage, and so such a patient may truly be said to be living over a volcano. The author states that the total extirpation of the prostate is a very severe operation when one considers the age and physical condition of the average prostatic, but the operation should be fairly successful in those who have not yet reached an advanced age and who are still vigorous. Still one should remember that opposed to this operation, as also in castration, is the fact that the sexual apparatus is mutilated to a marked degree, and this is often followed by much mental anguish. Sexual life is abruptly closed by this radical operation, and the author believes that it should never be performed until a vasectomy has been tried and proved itself a failure.

The Radical Operation for Congenital Inguinal Hernia in Young Children.—KAREWSKI (*Centralblatt f. Chir.*, 1902, No. 51) states that the remarks by Klemm in No. 46 of that journal convince one that the difficulties of isolating the spermatic cord are greatly increased when one cuts down only as far as the serosa and then allows the tunica vaginalis to escape from the innermost layer of the sac. In no case did a parenchymatous hemorrhage or an accident to the cord or vas deferens take place. The isolation of the cord can be undertaken without difficulty to the internal ring; at that point the peritoneum should be drawn together and sutured, and as a result one obtains a support which will only give way as the result of a severe strain. The Bassini and other radical operations do not seem indicated except in those cases of large scrotal hernia. This simple method of treatment has proved its worth in many cases, some of whom have remained perfectly well for periods of ten and twelve years. The advantages are that it can be performed in a few minutes, and the wound, which is well removed from the danger of urine contamination, is usually cicatrized in eight or ten days. The greater proportion of the author's cases lived under most unfavorable surroundings, but every case was followed by a good result. It is best during the first few days after the operation to dress the wound daily with iodoform, for which vioform may later be substituted.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Coal-tar Drugs in General Nervous Affections.—DR. F. SAVARY PEARCE prefaces his more detailed remarks by the statement that the coal-tar products have their physiological and pathological action in the main

through impression of the central nervous system, and act according as they affect the centres governing trophic nerves or as they control or depress vasomotor competency. He speaks of the danger of administering these drugs for the relief of pain, as in the headache of neurasthenia, without treating the disease, and quotes an interesting case of the drug-habit thus formed. He thinks the use of the coal-tar products has been overdone in cases of hysteria, and warns against their use in case of pain arising from brain tumor; also in typhoid with cardiac failure, and in meningitis, which he prefers to treat without the use of an analgesic. He advocates the use of antipyrin, however, in hyperpyrexia due to cerebral or other disease, in some cases of idiopathic epilepsy, and in combination with acetanilid to relieve the paroxysms of pain in tabes dorsalis. He considers one of the most valuable indications for the coal-tar products in nervous diseases to be in disease of the spinal cord, but thinks there is little need of these drugs in organic disease of the central nervous system.—*Therapeutic Gazette*, 1903, vol. xxvii., p. 9.

Aqueous Preparations of Menthol.—Dr. M. Cresantignes describes a method of obtaining an aqueous solution of menthol without the addition of the large amount of alcohol generally considered necessary to dissolve it. He has found it excellent as an antiemetic, having proved by administering it after ipecac that it controls the reaction of the latter. In vomiting of reflex origin he has found it excellent, and, except in cases of gastralgia, he has used it to inhibit the spasm. He has succeeded in obtaining a satisfactory aqueous solution of menthol by using the tincture of *quillaja saponaria*, used by druggists in making emulsions. His formula is as follows: Menthol, 5; tincture of quillaja, 500; distilled water, 101 to 2500. After the menthol is dissolved in the tincture glycerin is added, and then the water in small quantities, and shaken thoroughly. Two drachms constitute a dose. The amount of menthol in each dose is very small, but it is better to give it thus diluted, several spoonfuls at a time if necessary, as in more concentrated solution it is apt to burn. For external application he recommends the following formula, which he finds relieves frontal headache due to fever or other causes when applied as a wet compress for a few minutes only, and followed by a compress of fresh water: Menthol, 30; tincture of quillaja, 10; distilled water, to 155. As a gargle and mouth-wash he recommends the following: Menthol, 20; tincture of quillaja, 20; saturated aqueous solution of boric acid, 1000.—*Les Nouveaux Remedies*, 1903, vol. xix., No 1, p. 1.

The Influence of Some Modern Drugs on Metabolism in Gout.—Dr. William Bain says that the increasing attention paid to the observations in the constitution of the urine in gout has been partly directed to the relative preparations of the nitrogenous end-products. From the standpoint of therapeutics he has studied a number of newer drugs, particularly with reference to their effect on these nitrogenous end-products. The products studied were piperidin tartrate, lithium benzoate, piperizin, lysidin, urotropin, sidonal, and colchisal. With the main object of ascertaining the influence of the drugs mentioned they were administered to a case of chronic gout,

the patient being on a fixed diet during the investigation. With reference to his results he found that piperidin tartrate showed but slight influence on the excretion of uric acid, but that the rise after its discontinuance warrants the hope of more definite results if given in larger doses for a longer period. Lithium benzoate seemed to give absolutely no results; piperizin had a slight augmentive influence on the excretion of uric acid, but a more striking variation was the diminution in the aromatic sulphates. Lysidin gave a distinct increase in the uric acid excretion. Urotropin, so far as uric acid was concerned, gave no results. Sidonal gave the most marked advances in the uric acid excretion. Colchisal stood at the opposite end to sidonal, with a minimum excretion, and it is noted that the ethereal sulphates rose to a high point. The patient was of the opinion that he derived greater benefit from colchisal than from any of the other prescribed substances.—*British Medical Journal*, 1903, No. 2196, p. 243.

OBSTETRICS.

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An Unusual Case of Rupture of the Uterus.—In the *Monatsschrift f. Geburtshülfe und Gynäkologie*, 1903, Band xvii., Heft 6, KNAUER reports the following interesting and remarkable case: The patient was a multipara, admitted to the hospital in labor after an unsuccessful attempt to deliver by the use of forceps. On examination the condition of the abdomen and the patient's prostration and shock pointed to rupture of the uterus. The fœtal head was above the entrance to the pelvis, and the fœtal heart sounds could not be heard. The cervix was dilated and the membranes ruptured, and no serious injury to the uterus could be detected. The pelvis was not contracted. After craniotomy examination of the interior of the womb was made and the lower uterine segment was found apparently uninjured. In the left broad ligament there was a tumor as large as a fist close to the uterus. The tumor was apparently a hæmatoma. The pulse was small and frequent, although it was thought that internal hemorrhage was not proceeding. Cold was applied to the abdomen, and during the following night the patient vomited and the abdomen became distended. Five days after labor the patient died, her condition not justifying abdominal section.

Upon autopsy a hæmatoma was found in the left broad ligament, extending to the symphysis and dissecting up the peritoneum. This hæmatoma had ruptured, permitting free hemorrhage into the abdominal cavity. In the right side of the pelvis, near the acetabulum, was found a mass of hard lymphatic glands, tuberculous in nature. A rupture in the uterine muscle

was found without an injury to the outer serous layer covering the lower uterine segment. The tissues surrounding the uterus had been adherent at the location of the enlarged glands, and the disturbance incident to labor had torn the adhesions and had caused rupture.

Knauer also reports the case of a multipara who had a tedious labor, during which an elastic dilator was introduced to hasten delivery. Symptoms of premature separation of the placenta occurred, and the membranes were ruptured, the cervix dilated with Bossi's dilator, and craniotomy was rapidly performed. The patient collapsed after delivery, and soon died.

On autopsy numerous ruptures in the posterior wall of the uterus on the left side were found opening into the muscular tissue and through the adjacent tissue. The placenta had prematurely separated.

The writer also reports a case of chronic Bright's disease, with hypertrophy of the heart, hemorrhagic hepatitis, and infarcts in the uterus, with hemorrhage. Additional cases are cited to show that it is possible for hemorrhage to occur from the uterus without rupturing the peritoneal covering. Some of these cases are accompanied by premature separation of the placenta.

Artificial Rupture of the Membranes as an Aid to Labor.—GIBB (*Scottish Medical Journal*, April, 1903) calls attention to cases in which the presence of the amniotic liquid acts as an obstacle to the progress of labor. He describes a case of polyhydramnios in which labor remained at a standstill until the membranes were ruptured, when premature twins were born. He also describes the case of a multipara in labor with no water in front of the head and with delay in labor. On rupturing the membrane labor proceeded. He reports a case in which the lips of the cervix were very thick and the water in front of the head very scanty. Long delay occurred in labor until the membranes were ruptured, when the child was soon born. He also describes three cases of hemorrhage from partial separation of the placenta in which spontaneous delivery and recovery of the mother followed rupture of the membranes.

The writer recognizes the value of an amniotic liquid when it collects before the head in moderate quantity. Under these circumstances the membranes should not be ruptured; when, however, the amniotic liquid is present in excess or is deficient, labor will be expedited by rupturing the membranes.

Hysterectomy for Puerperal Septic Infection.—In the *Revue Pratique d'Obstétrique* of May, 1903, PINARD expresses his positive belief that hysterectomy is not indicated in puerperal septic infection. In a total of 26,952 cases of labor at term and abortion the septic mortality was one-fourth of 1 per cent. Hysterectomy was performed in two cases. One was septic after labor at term, and thrombophlebitis with suppuration in and about the uterus was diagnosticated. Hysterectomy was performed on the tenth day after labor. Autopsy showed suppurating pleurisy upon the left side, caused by a streptococcus infection. This had not been diagnosed.

The second case occurred after labor at full term attended by a midwife. In the absence of other signs the patient was believed to have multiple

abscesses in the wall of the uterus. Hysterectomy was performed, the cervical canal being cauterized and drainage employed. At autopsy it was found that suppurating phlebitis in the uterine and ovarian veins of the right side was present.

Pinard concludes that hysterectomy is not indicated in the treatment of puerperal septic infection. He excepts cases of suppurating fibroids or cases in which the placenta is adherent and cannot be removed, and cases in which the uterus has been ruptured during labor.

GYNECOLOGY.

UNDER THE CHARGE OF

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Gonorrhœal Peritonitis.—HUNNER and HARRIS (*Johns Hopkins Hospital Bulletin*, 1902, No. 6) report seven cases of acute diffuse peritonitis of gonorrhœal origin, with two deaths after operation. They have collected thirty-nine cases, of which twenty-four were operated upon, with a mortality of 20.8 per cent., the mortality without operation being 46.7 per cent. The mortality is especially high in children.

Cancer of the Vagina due to Mechanical Irritant.—MALY (*Zentralblatt für Gynäkologie*, 1903, No. 27) reports a case of extensive cancer of the vagina due to the long-continued pressure of a rubber ring pessary, and refers to six other similar cases mentioned in Veit's monograph on *Diseases of the Vagina*. The disease developed at the point of greatest pressure; that is, in the posterior fornix or just behind the symphysis in the case of a Hodge pessary, and on the lateral walls when a ring was used.

In the case reported an erosion first appeared, which healed when the ring was removed, and returned and became cancerous when the pessary was again worn. Uncleanliness seemed to be an etiological factor. In view of the rare occurrence of cancer of the vagina in patients wearing pessaries for a long time (in one instance nearly forty years) the writer infers that there must be but a slight predisposition to development of the disease from mechanical irritation. As regards the question of the origin of cancer from erosions, he offers the explanation that in the attempt at repair the formation of new tissue may become excessive, so as to destroy the equilibrium between epithelium and connective tissue.

Retroversion and Appendicitis.—NEUGEBAUER (*Zentralblatt für Gynäkologie*, 1903, No. 27) reports five successful cases in which appendectomy and Alexander's operation were performed simultaneously. In all the diagnosis

of appendicitis was made before operation. After shortening the round ligaments the incision on the right side was extended upward to the appendical region. The writer opposes a median incision in these cases on account of the difficulty of reaching and removing the appendix when situated behind the cæcum or buried in adhesions, and also the necessity of making an additional lateral incision for the purpose of drainage. It is assumed that the uterus and adnexa are perfectly movable.

Sarcoma of the Uterus with Extensive Thrombosis.—HENNICKE (*Zentralblatt für Gynäkologie*, 1903, No. 27) reports the case of a multipara, aged fifty-seven years, who entered the hospital with a large nodular uterus and a hard, irregular tumor filling Douglas' pouch. On opening the abdomen an intraligamentary growth was found, which corresponded in situation to the ovarian vein and could be traced upward as a thick cord in the direction of the kidney. The uterus and tumor were removed, the patient making a good recovery and having no evidences of recurrence a year after operation. The uterine neoplasm proved to be a spindle-celled sarcoma which had extended into both the uterine and ovarian veins. It was evident that the disease had also involved the adventitia and grown toward the lumen. The case is regarded as unique.

Treatment of Uterine Fibroid.—KANNEGISSER (*Journ. Akusch. i Shensk. bolesnej; Zentralblatt für Gynäkologie*, 1903, No. 27) reports 462 operations for fibroid in Ott's clinic up to January 1, 1902. The indications for operation were profuse hemorrhage, rapid growth, pain and pressure symptoms, and inability to work. Conservative work was done in 14 per cent. Supravaginal amputation was performed 85 times with 15 deaths; 210 vaginal operations were done with only 7 deaths; total abdominal extirpation in 75 cases was followed by 6 deaths. The writer infers from these statistics that total extirpation with vaginal drainage is preferable to supravaginal amputation except in cases in which it is necessary to shorten the operation on account of the weak condition of the patient. Abdominal section is to be elected only when it is impossible to remove the tumor per vaginam.

Effect of Ligation of the Uterine Vessels.—GUSSAKOW (*Journ. Akusch. i Shensk. bolesnej; Zentralblatt für Gynäkologie*, 1903, No. 27) draws these conclusions as the result of a series of experiments in rabbits: The uterus is normally nourished by the uterine arteries, and when these are ligated atrophy of both the endometrium and muscular tissue results. In the human subject double ligatures must be applied to both arteries on account of the free anastomoses between the vessels. It is also important to tie the arterial branches which extend outward from the uterine cornua and anastomose with branches of the ovarian.

No especial skill is required in the operation, which is preferable to all other palliative measures in the treatment of uterine fibroid as well as in cases in which a radical operation is not practicable.

Neuralgia due to Gonorrhœa.—KALABIN (*Zentrablatt für Gynäkologie*, 1903, No. 27) reports two cases of sciatica and intercostal neuralgia in

recently married women with acute gonorrhœal infection. In both instances the nerve pains appeared two weeks after the beginning of the discharge and continued for a month. The writer infers a direct etiological relation between the two conditions, possibly explained by irritation of the cerebral centre by gonococci.

Lymphatics of the Ovary.—POLANO (*Monatsschrift f. Geb. u. Gyn.*, Band xvii., Heft 4) conducted a series of experiments on dogs with the view of determining if obstruction to the flow of lymph in the minute vessels follows ligation of the main trunks. Great care was taken to isolate the afferent and efferent lymphatics in the meso-ovarium, so as not to include the blood-vessels in the ligatures. Six human ovaries were also injected and studied. The writer's conclusions are as follows: In the cortex the fine lymphatics are distributed around the periphery of the follicles, the tunica interna and granulosa containing none. In the corpora lutea and albicantia the lymphatics are found only in tissue derived from the tunica externa. A network of lymphatics surrounds the younger, deep-seated follicles, but these are absent in the neighborhood of the ripe superficial ovisacs. No lymph vessels could be demonstrated in the albuginea, but they became larger toward the zona granulosa, where larger lacunæ were seen, lined with endothelium. Nowhere could an intimate relation between the lymph and blood-vessels be detected.

No additional light was obtained by the examination of ovaries from the newborn and aged. In general the lymphatics of the ovary presented throughout the same capillary arrangement. A direct communication between these and the connective tissue and interspaces could not be proved. Neither in the human ovary nor in those of animals could it be shown that the bloodvessels entered lymph spaces. The theory of perithelia in the ovary must therefore be abandoned.

OPHTHALMOLOGY.

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The Relation and Treatment of Follicular Conjunctivitis and Trachoma.—ALGER, New York (*Medical Record*, January 24, 1903), calls attention to the relation between these two diseases—a matter of renewed importance now that the Government is excluding immigrants suffering from contagious eye diseases, and children similarly afflicted are being excluded from the schools.

The differential points in the diagnosis between follicular conjunctivitis and early trachoma given by most writers are absolutely useless for clinical purposes, and the confusion between these two diseases is readily explicable. Trachoma is rare among children. Most of those who are excluded from the schools for supposed trachoma do not have it, but a comparatively insignificant disease which threatens no serious mischief, and which recovers almost of itself. Follicular conjunctivitis is simply reaction to local irritation, which may be chemical, mechanical, or bacterial in its nature. The subjective symptoms are almost *nil*. If the lids be everted, small, round granules not larger than pinheads, arranged in parallel rows, like beads, are found upon the membrane adhering to the lids. In the looser tissue of the fornix they are much larger, resembling frog-spawn or grains of boiled sago. So far as these granulations go, the appearances are the same in trachoma. A diagnosis based thereon is mere guesswork. The essential feature of trachoma is hypertrophy of the conjunctiva. Upon this we must depend for the diagnosis until bacteriology finds a specific cause for trachoma, or the cornea begins to show characteristic involvement, pannus, etc., and the fibrotarsus of the lids becomes destroyed or distorted, giving rise to entropion, ectropion, etc. Trachoma is certainly a contagious disease. Its subjective symptoms are marked photophobia, feeling of sand in the eyes, purulent discharge, etc. In the treatment of simple follicular conjunctivitis the cause, as far as possible, should first be removed, whether chemical, mechanical, or bacterial. This involves frequent irrigations with a solution like boric acid, and with attention to the surroundings and physical condition of the child. The writer recommends swabbing the conjunctiva three times a week with a solution consisting of ichthyol, ♏xv; tr. iodi., ʒj; glycerin, ʒj. On alternate days an instillation of the following is made at home: Zinci sulph., gr. j; vin. opii., ♏viij; aquæ, fʒss. The roller forceps is a great time-saver, though some after-treatment is generally required.

The treatment of real trachoma is a very different matter. The author has used the above ichthyol mixture and has obtained results about as good as with other plans of treatment—not much better, not worse.

[This insistence upon the difference between two diseases which have certain appearances in common, but differ so greatly in gravity and results, is most timely. The sensational statements which have found their way into the newspapers as to the prevalence of trachoma among the school children in large cities, so far as they are based upon facts at all, are to be traced to the confusion into which the inspectors have fallen. The subject has been eagerly seized by the fomenters of race prejudices, with the result of adding to the burdens, already sufficiently heavy, of the defenceless poor of the large cities.—T. B. S.]

Results in One Thousand Consecutive Cataract Extractions.—MAYNARD, Calcutta (*India Medical Gazette*, February, 1903), in 1000 consecutive cataract extractions performed on 864 patients, of whom 136 had both eyes operated, obtained good results (vision from 6/6 to 6/36) in 89 per cent.; indifferent (vision poor, but sufficient to enable the patients to go about alone) in 5.7 per cent.; failures in 4.5 per cent. Of the failures 3.6 per cent. were due to sepsis, the others to intraocular hemorrhage, iritis in two cases,

iridocyclitis in one, and detached retina in one. Of these 45 failures 26 had been done with iridectomy and 19 without. The indifferent results (5.7 per cent.) were due to various causes, such as sepsis, opaque cortex, iritis, glaucoma, overripeness, mercurial cloudiness, vitreous prolapse, and so on. Of 351 patients from whom family histories were obtained, in 84, or nearly 24 per cent., parents and other relatives had had cataract. Diabetes was present in only 6 cases; all of these did well. The author does not believe in the connection between diabetes, which is very common in Bengal, and cataract. In 814 cases the average age was under fifty-three years—somewhat higher in males than with females—thus agreeing in the general belief that cataract comes on earlier in the tropics.

One hundred and eleven unripe cataracts were extracted, with 93 good, 9 indifferent, and 8 bad results (this leaves one unaccounted for). Sixty-three overripe lenses were extracted, with 57 good, 5 indifferent, and 7 bad results. Fourteen of these had prolapse of vitreous, 2 suppuration, and 1 each was lost from intraocular hemorrhage and retinal detachment. Tension was increased in 41 cases; of these 32 gave good results, 5 indifferent, and 4 bad. Tension was diminished in 90 cases; of these 77 gave good results, 8 indifferent, 4 bad, and 1 unknown. Vitreous prolapse occurred twice where tension was raised and six times where it was lowered. In no case was the tension altered beyond + or — 1.

Chloroform was used in 8 cases, cocaine in 910, and eucaine in 82. Eucaine was given up on account of the pain and increased hemorrhage it causes; otherwise it is an ideal anæsthetic.

The patients were prepared for operation by washing the face, forehead, and eyelids with soap and water, and then with sublimate solution (1 in 5000) the day before the operation. The eyelashes were cut and the conjunctival sac washed out with sublimate and a bandage applied. On the morning of the operation this procedure was repeated. In the last series the sublimate was replaced by biniodide, 1 : 10,000, with improvement as regards sepsis. The instruments were boiled and the dressings carefully prepared in the last 700 cases, with results nearly twice as good in these as in the earlier series. Atropine was used almost always both before and after the operation.

The appearances of the lens before operation are noted in 517 cases and compared with the condition of the capsule, cortex, and nucleus after removal, with the resulting vision. Milky-white lenses were found most often to have a tough or thin capsule (117 out of 129) and a brown or yellow nucleus. These gave the best results, and are the only kind in which it is advisable to omit an iridectomy. Lenses with glistening white sectors are of the same nature, and probably develop into this kind if left. Gray uniform lenses have often a "greasy" capsule (42 out of 110); *i. e.*, the cystitome cuts them as if scraping cold bacon. They often have a solid and glutinous cortex (21 and 46 out of 110) and generally a brown nucleus, making removal difficult, bruising the iris and requiring subsequent discission. These require a larger incision for extraction. Gray striated lenses resemble white striated lenses as regards cortex and nucleus, but their capsules are often "greasy," requiring large incisions and iridectomy, as do also brown lenses, which often have a greasy capsule and glutinous cortex as well as

cribriform lenses. These are not uniform gray lenses with transparencies and opacities giving the appearance of a veil. Black cataracts are rare, only 5 in the 1000. Their capsules were mostly "greasy" and tough. Iridectomy was done in all with resulting good vision, except in one case, in which it was only 1/60.

From measurements of the cornea and lens the lens diameter may be taken, roughly, to be a little more than two-thirds of the corneal horizontal diameter.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Milk and Infant Mortality in France.—A contribution to the *Annales d'Hygiene Publique*, September, 1902, by DRS. CH. GIRARD and F. BORDER, under this title, constitutes a terrible impeachment of the sanitary administration of French towns, in which it appears that the laws against the adulteration of milk must be absolutely dead letters. They do not hesitate to state that the milk as retailed is "not only wanting in the requisites of a passable article of food, but has undergone such countless sophistications that it has in many instances lost all alimentary value, and must be looked upon as a veritable poison." The climax seems to be reached at Lille and Roubaix, the municipal authorities of the former town admitting that the bulk of the milk sold had been passed through a separator and retained but 5 to 7 grammes of butter per litre, or, as we should say, 0.5 to 0.7 per cent. of fat. Of 694 samples examined in the municipal laboratory at Lille 20 per cent. had less than one part and 35 per cent. between one and two parts per one hundred of fat, only 16, or 2.3 per cent., being really good.

In the year 1897 or 1898 the mayor had secured the passing of a by-law fixing the standard of fat at 2.5 per cent., but it provoked violent opposition, and was soon rescinded as an abuse of power and arbitrary interference with the liberty of the individual. The inquiries carried out by our authors seem to have had the effect of "waking up" the authorities, for the regulation was revived, with the provisos that milk might be sold with 1.5 to 2.5 per cent. if distinctly labelled "lait lécrémé," and with less than 1.5 per cent. if marked "lait pauvre." As a result of the vacillation of the authorities the percentage of samples retaining the full percentage of 3.5 of fat fell from 15 to 6.6 in 1900, and rose again to 17.4 in 1901.

Drs. Girard and Border extended their inquiries into the milk-supply and infant mortality to every town of importance in the country, but met with little assistance, if not with actual obstruction from the local authorities. But the value of their statistics is greatly impaired by their adherence to the obsolete and unscientific practice of calculating the deaths of infants under one year of age on those of persons of all ages—a ratio that tells us nothing unless we know the relative numbers of the living at the respective ages.

Only in the case of Lille do we get a glimpse of the truth. There the number of births in the year 1900 was 6228, and of deaths under one year of age was 4984. Many of these, however, being those of children born in 1899, the surviving infants at the end of the year 1900 were 4766. Tuberculosis and "athrepsie," or diarrhœa and wasting diseases of children, each account for 15 per cent. of the total mortality at all ages in France; while of those of infants under a year from 20 to 60 per cent. are ascribed to "athrepsie"—in other words, to improper feeding—probably the heaviest preventable infant mortality in any community claiming to be a part of the civilized world.

The Etiology of Acute Rheumatism and Allied Conditions.—BEATON and WALKER (*Lancet*, January 31, 1903, p. 237) have isolated a micrococcus from 8 cases of acute rheumatism, 3 cases of chorea, and 4 cases of acute endocarditis in rheumatic subjects which they regard as specific in these infections. The organism resembles the streptococcus morphologically and culturally, but differs from the ordinary streptococcus inasmuch as it grows freely in filtrates of old bouillon cultures of the ordinary streptococcus of human origin. According to Marmorek this growth would rule out the ordinary streptococcus, which he has found will not grow in such filtrates. The organism when injected intravenously into rabbits gives rise to fever and wasting, monoarthritis, polyarthritis, pericarditis, endocarditis, septicæmia, and death. The writers regard their organism as identical with those described by Triboulet, Wassermann, and by Payne and Poynton.

On Active Immunization Against Plague, Cholera, and Typhoid Fever.—BESREDKA (*Annales Pasteur*, 1902, vol. xvi. p. 918) has compared carefully the relative value of active and passive immunity in plague, cholera, and typhoid, and proposes a new method of active immunization which does away with the untoward effects of active immunization as described by Haffkine.

The inoculation of the specific antisera in the diseases mentioned will give rise to an immunity which is immediate, but very temporary, lasting only from eight to fifteen days. Inoculations with killed cultures, according to the method of Haffkine, give a lasting active immunity, but, unfortunately, is preceded by an incubation period of from one to two weeks, during which time the animal is more than usually susceptible to infection; in addition, the localized abscesses, loss of weight, and malaise make this method far from ideal in the human being.

To obviate this difficulty Calmette and Salembini injected a mixture of antiserum and killed organisms. The untoward effects are obviated, but the duration of the immunity is not greater than with the serum alone. The author has succeeded in creating an active immunity by suspending the living

cultures in the antiserum and then washing the agglutinated organisms in normal salt solution until all excess of serum is removed. Such a mixture gives no violent reaction, and at the same time an immunity is brought about which is present within twenty-four hours, and which lasts as long as the active immunity of Haffkine. The vaccines which are prepared in this manner will also retain their immunizing power for at least several months. —F. P. G.

Peritonitis in Typhoid Fever without Perforation, with a Report of One Case Caused by the Bacillus Typhosus and Another Simulating Acute Appendicitis.—YATES (*American Medicine*, 1903, vol. v. p. 700) discusses the subject of peritonitis in typhoid fever without perforation, and adds two such cases to the literature. The author finds from a survey of some of the extensive statistical records of typhoid fever that this complication occurs in about 1.7 per cent. of the cases. Its symptomatology does not differ from that of the perforative type. The origin of the peritonitis in some of the cases was traced to a rupture of the mesenteric glands, but in no instance did infarctions of the spleen appear to give rise to the condition. The typhoid bacillus is usually the direct cause of the peritoneal inflammation. From the author's study of his own cases he concludes that in the one fatal case the peritonitis was caused by a direct extension of typhoid bacilli through the ulcerated intestinal wall. The deep ulcers and meteorism were thought of as predisposing factors. In certain areas thrombi were found in the vessels of the intestinal wall. Among the thrombi were some composed of agglutinated red blood corpuscles. Experiments were made to determine whether the typhoid bacillus, or the product of its growth in cultures, was capable of agglutinating human red blood corpuscles *in vitro*. These experiments demonstrated definitely that the typhoid bacillus generated agglutinin which had a specific action upon human blood.

The Agglutinating Properties of Bile.—CANTANI (*Zentralblatt f. Bakt. u. Parasit.*, 1903, Bd. xxxiii. p. 731) reports experiments made to determine the agglutinating power of bile from normal animals and from immunized animals upon different varieties of bacteria. It was found that bile from normal dogs, guinea-pigs, oxen, and rabbits did not agglutinate any of the bacteria used, among which were B. coli, B. typhi, B. influenza, staphylococci, and streptococci. Animals were inoculated with these various organisms and the bile tested for agglutination during the stage of acute infection, but it was found that at this time no agglutination occurred. If the animals recovered from their infection, the bile occasionally showed some slight power to agglutinate that particular variety of bacterium which had produced the infection. On the other hand, the bile of animals highly immunized against a specific organism was capable of agglutinating that organism rapidly and in high dilutions. The agglutinating power of the bile, however, never exceeded or even reached that of the serum. From these observations the author concludes that if the agglutinins are present in the blood serum only in small amount, they do not appear in the bile; and it is only when they occur in great quantity in the blood that they pass over into the bile itself.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

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Significance of Nitrites in Water.—The opinion of a number of recent writers to the effect that the significance of nitrites in water is sometimes overrated receives additional confirmation from a study of the subject by Dienert (*Révue d'Hygiène* April 20, 1903), who concludes as follows: 1. Nitrites may be found in the subsoil in the absence of pollution. 2. Pollution is not always followed by the formation of nitrites. 3. It is necessary to take certain precautions in testing, for nitrites may be formed during transportation if the temperature is sufficiently high. The test should be made on the spot at the time of taking the sample. 4. Judgment of potability should not be based upon the presence of nitrites.

The Campaign Against Malaria.—It is stated by KOCH (*Zeitschrift für Hygiene und Infectionskrankheiten*, xliii., May 22, 1903, p. 1) that when he first turned his attention to the study of malaria he soon became convinced that the best plan of campaign was to seek out the bearers of the parasite and destroy it in their systems with quinine, paying no attention to the specific mosquito; for the parasite is to be found only in the human body and the anopheles mosquito, which latter, in the absence of infected persons, cannot generate it themselves. In an experiment at Stephansort he restricted the work of eradication on these lines and with great success. Prosch (*Ibidem*, p. 5) conducted a similar experiment in the islands of Brioni, and the work was carried out so thoroughly that whereas, prior to the experiment, whoever came to Brioni, even for a short visit, during the fever season was almost sure to contract the disease, and this condition had been the case beyond the memory of man. At the end of two years' seeking out those who harbored the parasite and treating them continuously with quinine, the disease was practically eradicated; and 170 men who settled on the island free from the disease remained more than a year without a single seizure. Bludau (*Ibidem*, p. 67) carried on a similar campaign at Punta Croce, on the island of Cherso, with equally favorable results; and Vagades (*Ibidem*, p. 83) another in Franzfontein, German Southwest Africa, also successfully. Bludau found the parasites chiefly in children, of whom but four out of thirty-two failed to yield them. Vagades found in his first examination that 68 per cent. of the natives were infested, and in his later examinations the proportion increased to 75 per cent. Among the surrounding tribes, from 28 to 71 per cent. were found to be carriers of the parasite. After a year's work on Koch's lines a very great improvement was observed, for during the next twelve months but fifteen cases of the disease were discovered, and four of these were imported cases.

The genesis of winter outbreaks of malaria is explained by Jancso

(*Deutsches Archiv für klinische Medicin*, lxxvi., p. 474), who tells of an outbreak of the æstivo-autumnal type which occurred among the attendants and patients of a hospital during winter weather, with ice and snow on the ground. Infected mosquitoes may be present in buildings even when the outside temperature is inimical to their activity, and in this particular instance the distributing agents were infected laboratory specimens which escaped from their cage and made their way to the wards and other parts of the hospital, where they bit a number of inmates, who came down with the disease. The mosquitoes were caught and examined and were found in all cases to contain the sporozoites.

Toxicity of Liqueurs.—The French Government requested the Academy of Medicine, some time ago, to make a study of the general question of the toxicity of liqueurs and to submit a list of those essential oils employed in their manufacture which may be considered as dangerous and another of those which are harmless. On March 10, 1903, the committee to whom the subject was referred, consisting of Laborde, Brouardel, Lancereaux, Cornil, Magnan, Pouchet, Motet, and Joffroy, reported their conclusions and submitted a list of oils which should be absolutely prohibited in the manufacture of drinks, because of their dangerous nature. This list included: oil of wormwood (absinthe), oil of rue, oil of gaultheria, oil of staranise, oil of hyssop, and oil of angostura. In a second list of objectionable oils, but not sufficiently objectionable to warrant absolute prohibition, are mentioned the volatile oils of clove, mace, nutmeg, peppermint, and a number of others. The Academy voted that all volatile oils, natural and artificial, and the various extractives which are incorporated with alcohol or wine, make drinks that are injurious and dangerous, the danger being due to the oils and alcohol. It calls particular attention to the danger of drinking them before meals, when their absorption is most rapid and their poisonous properties most active. It was also voted to recommend and to urge that measures be taken to diminish the sale of such drinks.

The Sanitary Significance of Tin in Foods.—As the result of extensive investigation of the subject of tin in foods, PROFESSOR K. B. LEHMANN (*Archiv für Hygiene*, xlv., p. 88) comes to the following conclusions: 1. Acute, but usually slight, disturbances of digestion may be caused by the use of foods which contain tin in large amounts in soluble form. Especially suspicious are old preserves containing malic and tartaric acids, if large amounts are consumed at one time. The number of cases of undoubted poisoning due to such foods is very small. 2. Ordinary canned meats and vegetables which are not acid or are only slightly acid rarely appear to cause acute poisoning; at least, in spite of the enormous use of these, no case is known. When one hears of "acute tin poisoning," one should think of spoiled food, and tin should not be blamed until all other explanations fail. 3. Cases of chronic poisoning from amounts of tin such as can be taken up on long contact (4 to 6 milligrammes per kilo and day) have never been observed. In experiments with cats, 10 to 14 milligrammes per kilo daily for a year to a year and a half did not appear to be markedly injurious. 4. Idiosyncratic susceptibility to tin is a theoretical possibility, but proof is lacking that such exists. 5. No special

precautions appear to be necessary in the use of canned foods except with those which contain large amounts of malic acid or tartaric acid. Such should be put up in glass, porcelain, or wood. Further investigation with regard to acetic, lactic, and other acids is desirable. 6. In spite of the small danger to be anticipated from tin, it would be fortunate if a process of packing could be discovered which would effectually obviate the necessity of ingesting that metal with canned foods.

On the other hand, DR. PATRICK MANSON (*Journal of Tropical Medicine*, October 1, 1902) asserts that in the tropics cases of peripheral neuritis have been recognized as resulting from the ingestion of tin.

Bacteriological Condition of Milk After Treatment with Borax.—DR. A. P. F. RICHTER (*Archiv für Hygiene*, xliii., p. 151) has investigated the behavior of milk with borax, especially with reference to the checking of the growth of bacteria and of the coagulation of milk. He found that slight variations in the time of coagulation were due to differences in the freshness of milk and in the temperature of the place of storage, this ranging from 15° to 25° C. He succeeded in preventing coagulation permanently only after the addition of 4 per cent. of borax, but this amount of the drug caused such a disagreeable taste as to make the milk useless. His conclusions are as follows: 1. The development of bacteria seems to be furthered after a very short time, and this is due, perhaps, to the fact that the addition of borax facilitates the dissemination of colonies of bacteria. 2. The growth of *oidium lactis* is hindered considerably by borax. 3. The same is true of *B. acidi lactici* (Huppe) and *B. acidi lactici* (Günther). 4. The liquefying and other bacteria of putrefaction are not hindered in their growth by borax, but after a time they perish spontaneously. 5. In the first few days Hueppe's lactic acid bacteria are present in greatest number, especially in untreated milk, but they are succeeded later on by those of Günther. The greatest number of colonies occurs on the second and third day. From the sixth to the eleventh day there is a considerable diminution in the number of colonies in pure milk as well as in boraxed milk. 7. The surviving cocci were almost exclusively air cocci, chiefly *micrococcus candicans* (Flügge).

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